ROLE OF ROUTINE SCREENING FOR GESTATATIONAL DIABETES MELLITUS WITH GLUCOSE CHALLENGE TEST IN ANTENATAL PATIENTS

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**ROLE OF ROUTINE SCREENING FOR GESTATIONAL DIABETES MELLITUS WITH GLUCOSE CHALLENGE TEST IN ANTENATAL PATIENTS"** is a bonafide record work done by **Dr. CT. SUMATHI** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for M.D Branch II – Obstetrics & Gynaecology.

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DECLARATION

I **Dr. CT. SUMATHI** solemnly declare that the dissertation titled **"ROLE OF ROUTINE SCREENING FOR GESTATIONAL DIABETES MELLITUS WITH GLUCOSE CHALLENGE TEST IN ANTENATAL PATIENTS"** has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of M.D degree Branch – II (Obstetrics & Gynecology) to be held in March 2010.

Place : Madurai **Dr. CT. SUMATHI**

Date :

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CONTENTS

INTRODUCTION

Diabetes mellitus, a clinical syndrome characterized by deficiency or insensitivity to insulin and exposure of organs to chronic hyperglycemia is the most common medical complication of pregnancy.

Preexisting diabetes affects approximately 1-3 pregnancies per 1000 births.

Gestational diabetes is defined as carbohydrate intolerance of variable severity with onset or first recognition during pregnancy. This definition applies whether or not insulin is required for treatment.

GDM complicates 3-4% of all pregnancies globally and 90% of these cases are definitely associated with a significantly increased maternal and perinatal morbidity. All complications associated with GDM are potentially preventable with early recognition of GDM, intense monitoring and proper treatment.

Moreover in view of the high prevalence of diabetes mellitus and its early onset among Indians, all pregnant women should be screened for GDM. Hence an appropriate method for screening of GDM has been much emphasized.

The importance of GDM is that two generations are at risk of developing diabetes in the future. Women with a history of GDM are at increased risk of future diabetes, predominantly type 2 diabetes as are their children. Besides any abnormal glucose intolerance during pregnancy also has adverse fetal outcome. Increasing maternal carbohydrate intolerance in pregnant women without GDM is associated with a graded increase in adverse maternal & fetal outcomes.

India falls under moderately high risk group and hence need to undergo universal screening. Hence this study is undertaken to know the incidence of GDM, need for universal screening & validity of the screening test.

CARBOHYDRATE METABOLISM DURING PREGNANCY.

Pregnancy alters carbohydrate metabolism but adaptation normally occurs without adverse effect on the mother or fetus. In some the maternal response to these changes is abnormal which without careful management would lead to increased fetal risk.

Pregnancy is diabetogenic. This is due to the increase in insulin resistance that occurs during gestation. Other reasons are increased lipolysis and alterations in gluconeogenesis.

DIABETOGENIC EFFECTS OF PREGNANCY.

1. Insulin Resistance

- Production of human placental lactogen
- Increased production of cortisol, estriol and progesterone which have anti-insulin effects.
- Increased insulin destruction by renal and placental insulinases.

2. Increased lipolysis

The mother utilizes fat for her caloric needs and saves glucose for fetal needs.

3. Changes in gluconeogenesis

The fetus preferentially utilizes alanine and other amino acids, depriving the mother of a major gluconeogenic source.

As a result of the physiologic changes of pregnancy the normal fasting blood sugar is 65±9 mg/dl. The mean non-fasting blood sugar level is 80±10 mg/dl.

During the fed state.

During the first few hours glucose absorbed from the gastro intestinal tract provides for the metabolic needs of the brain and other organs. The absorbed glucose in excess of these needs in used to rebuild fuel reservoirs in liver, muscle, fat and presumably in other tissues, synthesis of glycogen and triglycerides takes place. There is facilitated anabolism.

During the fasted state.

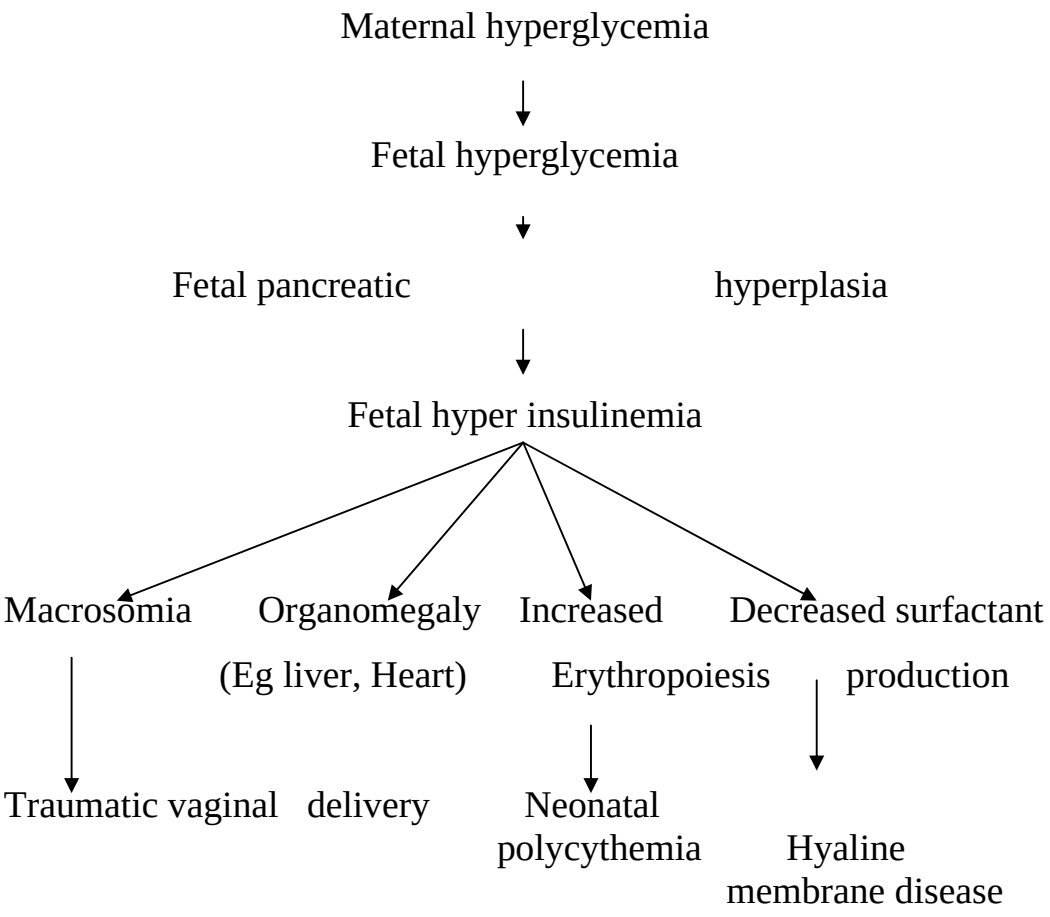
The pregnant woman changes rapidly from a post prandial state characterized by elevated and sustained glucose levels to a fasting state characterized by decreased plasma glucose and amino acids such as alanine. During fasting the plasma concentrations of Free fatty acids, Triglycerides and cholesterol are higher. This pregnancy induced switch in fuels from glucose to lipids is known as

accelerated starvation, certainly when fasting is prolonged in the pregnant woman these alterations are exaggerated and ketonemia rapidly appears.

So, there is facilitated anabolism in the fed state and accelerated starvation in the fasted state which characterize the maternal fuel adaptive changes during pregnancy.

Fuel Metabolism in Diabetic Pregnancy and Gestational Diabetes

Pederson's hypothesis



Blood Glucose and insulin relationship in the mother and fetus

In the normal pregnant woman there is a continuous demand by the fetus for glucose as an energy substrate and it crosses the placenta by facilitated diffusion. During fasting maternal glucose may fall significantly but in diabetes glucose levels are usually maintained and may be high without insulin.

The plasma glucose concentration of the fetus follows that of the mother closely, resulting hyperglycemia stimulates hypertrophy of fetal pancreatic islet cells resulting in increased insulin release which results in macrosomia and organomegaly due to glycogen synthesis, increased protein synthesis and deposition of fat.

Effect of pregnancy on Diabetes.

The considerable effects on carbohydrate metabolism particularly the lowered renal threshold for glucose and the diminishing sensitivity to insulin as pregnancy advances render the control of diabetes more difficult, so more insulin needed to achieve metabolic control. Poor control increases the incidence of maternal and fetal complications and is the single most important factor influencing the outcome of pregnancy. Careful plasma glucose control is mandatory because of the adverse effect of hyperglycemia

and ketosis on the fetus. This is difficult and needs special attention in early pregnancy when nausea and vomiting are common or when infection of any kind occurs and also during labour.

Other effects of pregnancy on diabetes are progression of diabetic retinopathy, worsening of diabetic nephropathy, increased risk of death for patients with diabetic cardiomyopathy.

Effect of Diabetes on the mother.

1. Spontaneous abortion

   Due to poor glycemic control in Ist trimester.

2. Monilial vaginitis and vulvitis
3. Pre eclampsia

   Affects 10-25% of all pregnant diabetics

4. Infection

   Urinary tract infection, high incidence of chorioamnionitis and post partum endometritis

5. Poly hydramnios
6. Post partum haemorrhage
7. Caesarean section

   High incidence in pregnant diabetics

Effects of diabetes on the fetus

1. Congenital abnormalities

Frequency of congenital abnormalities is increased in women with poorly controlled type I diabetes. The commonest are those of cardiovascular, skeletal and central nervous system.

2. Macrosomia.

Incidence of fetal macrosomia is increased in women with gestational and type II diabetes

3. Hyaline membrane disease.

Fetal hyper insulinemia may delay the maturation of surfactant production systems and particularly the synthesis of phosphatidyl glycerol and phosphatidyl choline. Insulin interferes with normal timing of glucocorticoid induced pulmonary maturation in fetus.

4. Unexplained fetal death.

Hyperglycemia mediated chronic aberrations in transport of oxygen and fetal metabolities may account for unexplained fetal deaths.

Osmotically induced villous edema led to impaired fetal oxygen transport. Fetal death may be due to placental insufficiency in association with severe preeclampsia.

5. Hypoglycemia
6. Hyperviscosity syndrome
7. Hypocalcemia
8. Apnoea and bradycardia
9. Traumatic delivery

Diagnosis of gestational diabetes at the earliest becomes very significant as early diagnosis and management can reduce the maternal and fetal complications to a great extent.

AIM OF STUDY

1. To find the incidence of abnormal blood sugar and gestational diabetes mellitus (GDM) by routine screening with 50 gm glucose challenge test in antenatal patients.

2. To evaluate the sensitivity and specificity of glucose challenge test and Random Blood glucose test.

REVIEW OF LITERATURE

HISTORICAL PERSPECTIVE:

In 1824 Bennewitz published the first case report of gestational diabetes. This formed the basis of his MD thesis 'Diabetes Mellitus: A symptom of pregnancy'. In addition to the classic symptoms and signs of thirst, polyuria and associated glycosuria, he described the death of a macrosomic fetus due to impacted shoulders. The patient's diabetes resolved completely after delivery, but recurred in two subsequent pregnancies.

The most comprehensive review of diabetes in pregnancy in the pre-insulin era was, however, published in 1909 by J.Whitridge Williams, of the John Hopkins University in Baltimore. He was careful to distinguish between physiological glycosuria and true diabetes and presented a review of 57 pregnancies between 1874 and 1909 involving 34 woman. The maternal mortality in this series was 25% and only 51% of woman gave birth to a living child.

In 1921 Frederick Banting and Charles best with the help of skilled chemist J.B.Colip found out the therapeutic active insulin.

In 1922 Banting received a Nobel prize. Since then this metabolic disorder has been studied extensively.

Screening for Gestational Diabetes should be universal (Hrish et al 1992). 2,561 women subjected to Glucose Challenge Test (GCT). In this study 470 were screen positive and they were subjected to 3 hour OGTT (O 'Sullivans Criteria). 80 women were diabetic and strict control was ensured among them and there was no significant difference with women of this group and with normal pregnant women.

Hence screening should be universal. In a study by Hughes et al and Agarwal et al 1995[7] found that selective screening would have failed to detect 43% of gestational diabetes 28% of them would have required insulin. Patients with GDM are at increased risk for macrosomia (26% Vs 11%) LSCS (37% Vs15%) shoulder dystocia (90% Vs 2%).

Screening should be done early as this factor reduces perinatal mortality and morbidity (Semmler-K, Semmler-S and [62] Steindel et al 1990). If this metabolic complication is determined and treated in a later phase of pregnancy there is a higher rate of complications. In their study PIH was 29.7% preterm labor was 21.8% LSCS rate

23.8% perinatal mortality 2.9% congenital anomalies 5-9% and macrosomnia 32.7%, the effect of glucose intolerance on pregnancy is so high that a screening measure was undertaken just to assess knowledge of Diabetes during pregnancy and was called DPKS (Diabetes in pregnancy knowledge screen) spirito et al and Ruggeiro et al 1990)[66]

[10]Coustan et al 1991 has drawn attention to an entity called Gestational Diabetes and it can be understood in terms of risk to the pregnancy and or risk to the mother. As various criteria are used in various parts of the world a pregnancy specific criteria is to be used (Berger et al 1990). In this study he has stated that glucose intolerance by glycosuria is unreliable and should be replaced by blood sugar (Screening), in the 24th to 28th week of gestation. This detects 3% of patients with glucose intolerance. High risk patients ideally should have their glucose checked earlier to conception, at 24 weeks to 28 weeks and also in 32nd to 36th week. Edelburg[69] and Philip son et al in 1991 in their study have diagnosed 66% of diabetes in I trimester in a high risk population with 50g GCT. However Stephenson et al 1991[22] has done a critical review and has found only macrosomia is consistently associated with GDM. The

reference standard and the OGTT are problematic in that there are no standardized testing procedures or definitive criteria for diagnostic interpretation. There is insufficient data to justify routine screening for GDM. However GDM is a challenge for the future and glucose intolerance develops in women unable to compensate for the metabolic changes incurred by pregnancy (Heigh et al 1992). Hence agreed criteria has to be followed, Screening for GDM can be done by estimation of blood or plasma glucose, Glycosylated hemoglobin, glycated proteins and even triglycerides (Knopp and Magee et al 1992)[41].

Screening for GDM to be done using a 50g glucose without prior patient preparation. One hour later venous blood to be drawn for glucose estimation and is called the glucose challenge test (GCT O 'Sullivan' et al) Threshold for further testing may be chosen based on the goal of the screening programme either to maximize sensitivity by using a 130mg/dl or to increase the specificity at the sacrifice of some sensitivity by using a 140mg/dl cut off (Coustan et al 1993[14]). [25]Gabbe et al (1991). In his study concluded that a plasma glucose level obtained 1 hour after a 50g GCT is the best GDM screening test. Khan et al[40] (1991) in his study did a 75g GCT

and plasma glucose was determined 2 hours later. Women who had abnormal screen earlier had the test repeated at 28-32 weeks gestation. Normal screen with a risk factor was 8.6% and subsequent follow up revealed 3.2% of GDM and 1.9% of impaired GTT. The advantage in using this 75g is that patient with plasma value greater than 170mg need not undergo repeat GTT.

[70]Lindenbaun et al and cohen et al in 1990 have compared values from capillary blood specimen obtained by means of a reflectance meter with that of venous sample after GCT and have found out that 90% of patients will not require lab studies which results in cost savings. Scheduling patients for OGTT was easy and women were most impressed.

Screening has also been done with fasting plasma glucose values and OGTT[44]. Mamsen et al 1990 has screened women with fasting plasma glucose levels. If the values were > 4 mmol/1 75g OGTT was performed and the incidence of GDM was 1.2% and if fasting plasma glucose is used one should measure after 31st week of gestation as most cases of abnormal OGTT occurs at that time. [59]Sacks et al 1922 has used fasting plasma glucose and has found out that this assay performed better than one hour GCT.

[8]Coustan et al 1991 has reviewed all the strategies and has ultimately recommended universal screening with a 50g GCT[16]. Chua et al and Rotham et al 1993 have performed a study with 50g GCT to establish the sensitivity and specificity and if the threshold of 140mg/dl is used diagnostic yield fell to 25.4%. There is a progressive increase in sensitivity if GCT was performed after 24 weeks without significant increase in specificity[52]. Neiser et al and constan et al 1992 have repeated GCT for women who had values greater than or equal to 130mg/dl after an average of 4-6 weeks and have repeated OGTT for women who had only one abnormal value in OGTT after an average of 4-6 weeks. Of this 34% turned out to be GDM patients. Hence even one abnormal value on GTT denotes a significant risk for the development of GDM, Watson[73] et al in 1989 has rescreened screen –ve patients at 34 weeks and 8% turned out to be screen +ve at 34 weeks.

Huisman and Dozy observed that HbA_1 which is the glycosylated fraction of Glycosylated haemoglobin was increased in diabetics. In Diabetes Mellitus the level of HbA_1C would be proportional to the integrated blood glucose level in the previous 7 to

8 weeks a period approximating to the half-life of the average red blood cell shown by Gabbet et al in 1977.

HbA_1C is formed by nonenzymatic glycation of hemoglobin and is dependent on the mean plasma glucose concentrations and the life span of the red cell. Normal non pregnant level is 5.7% and in gestational diabetes its 8.8%.

Disadvantages of HbA_1C is that both high and low values have been reported in chronic renal failure and the level is significantly reduced in patients with reduced red cell life span e.g. anemia & hemolytic anemia's and above all is costly. Hence measurement of blood HbA_1C is only an adjuvant.

Measurement of glycosylated blood proteins is established as a means of assessing long-term glucose control in DM.

Different methods of estimation of glycosylated serum proteins were developed and one such assay is the fructosamine assay (Johnson et al in 1987). [71]Glucose first condenses non enzymatically with the terminal amino group of the serum proteins to form an unstable aldimine. This under goes an Amodori rearrangement to form a more stable ketoamine. This ketoamine is usually termed fructosamine to its structural similarity to fructose.

Serum fructosamine values correlate closely with those of glycated proteins (Lyold et al 1985) and are easily reproductive (Baker et al 1985) [37].

In 1983 Roberts et al Baker et al designed a study to assess serum fructosamine as a screening test for the detection of gestational diabetes.

However in a study by Roberts et al 1990[58] concluded that glucose load had a sensitivity of 81% in diagnosing GDM when compared with 50% for fructosamine. Hence he concluded it's not an useful screening test. Fructosamine shows potential as an objective marker of short term control in evaluating the maternal glycemic state. (Cafalu et al & chester etal 1990)[12]. Fructosamine has a limited value as a screening test for GDM particularly for the mild form of glucose intolerance (nasrat et al & 1991)[35].

On the contrary (Narayanan et al 1991) an ideal lab test should accurately reflect short term Glucose changes. An objective strategy for lab monitoring of GDM should include assays such as fructosamine and even the less sensitive HbA_1C assay.

Fructosamine achieved 77.3% specificity and 79.4% sensitivity for diagnosis of GDM compared to GIT. (Haughes,

1995)[38]. As a screening test for GDM the role of fructosamine remains controversial with conflicting claims made by various investigators.

American college of obstetricians and Gynaecologist (ACOG technical bulletin 1986) has recommended screening for gestational diabetes using 50g gms/1hour GCT for all pregnant women aged 30 years or older and for women with risk factors. Coustan et al (1989) found that current ACOG recommendations resulted in sensitivity of only 65% and universal screening using a threshold of 140 mg/dl at 24-28 wks as recommended by second International Workshop (Diabetes 1985) had a sensitivity of only 90% Kini et al (1996) opined that 50 gms GCT should be repeated in third trimester as it yields a large no of gestational diabetics. We confined our study to single screening test at 24-28weeks as per current recommendations.

American Diabetes Association (ADA) recommends two step procedures for screening and diagnosis of diabetes in selective population. Compared with selective screening, universal screening for GDM detects more cases and improves maternal and off spring prognosis.

Another area of concern is that among ethnic groups in South Asian countries the Indian women have the highest frequency of GDM. Hence universal screening during pregnancy has become important in our country.

The incidence of gestational diabetes varies between 3-12% depending upon the population sample and the diagnostic criteria (carpenter 1982). Compared to European women, prevalence of gestational diabetes has increased eleven fold in women from the Indian sub continent (Dornhurst 1992). In a study conducted by Bhattacharya, Awasthi (2001) in pregnant women, overall incidence of gestational diabetes was 3.07%. Among the Indian workers, Maheswari et al (1989) and kumar et al (1993) found the incidence of gestational diabetes to be 4.9% and 5.5% respectively.

DIAGNOSIS

Early detection which aids in timely intervention is very important in pregnancy complicated by this metabolic disorder.

HOW TO DETECT EARLY

Unfortunately as onset of gestational diabetes has no reliable signs or symptoms this can be detected only though the use of laboratory test which we call screening tests.

SCREENING FOR DIABETES DURING PREGNANCY

The justification for screening is the increased risk of perinatal death amongst women who develop an abnormal GTT in pregnancy. If screening is to be effective it must be comprehensive. It should be simple, reliable, cheap and easy to interpret. Glycosuria alone is not significant.

WHOM TO SCREEN?

An area of controversy is whether screening for diabetes during pregnancy should be routine or if it should be limited to patient at risk for diabetes during pregnancy.

RISK FACTORS REQUIRING DIABETIC SCREENING (ARIAS)[23]

1. Obesity
2. Positive family history of diabetes (Sibling or parent)
3. History of still birth, intra uterine death.
4. History of delivery of a large infant (>4000g).
5. Glycosuria
6. History of unexplained neonatal death

7. History of congenital anomaly, prematurity, pre-eclampsia poly hydramnios, traumatic delivery with associated neurologic disorder in the infant.
8. Poor obstetric history
9. Chronic hypertension
10. Recurrent severe moniliasis & urinary tract infection
11. Age 30 years
12. History of gestational diabetes or impaired glucose tolerance in a previous pregnancy.

About 40-60% of women with GDM have no demonstrable risk factor. So screening should be universal. If only high-risk patients are screened, approximately 35% of gestational diabetic patients will not be discovered.

WHEN TO SCREEN?

Gestational diabetes typically occurs in the later half of pregnancy and has no effect on embryonic growth and thus is not a cause of congenital defects (Joselin). Gestational diabetes is a problem of the third trimester and the late second trimester. Hyperglycemia commencing during the second trimester results in behavioral changes while that occurring during the third trimester

causes only anthropometric changes in the fetus. American diabetes association suggests that all women be screened between gestational weeks 24 and 28. Patients at high risk may have the test earlier at first booking but if negative they should have the test repeated between 26 and 30 weeks. (DESWEIT)

SCREENING FOR GDM

Urine tests - Glycosuria

Blood tests

Fasting plasma glucose.

Random Blood glucose.

50 gm glucose challenge test.

75 gm glucose tolerance test.

Fructosamine assay.

Glycosylated Hb.

Glycosuria

The traditional method of waiting for glycosuria to appear has a low pick up rate and as such is of limited value. Sutherland et al found that 11% of an unselected obstetric population of 1418 women had glycosuria at some time, but fewer than 1% of those with glycosuria had an abnormal GTT.

Non challenge Blood glucose tests.

Non challenge blood glucose tests involve measuring glucose levels in blood samples without challenging the subject with glucose solutions.

Fasting plasma glucose .

Sacks et al and Daniele at al have observed that measuring FPG is an easier screening procedure and suggested a cut off value of 95 mg/dl for GDM. Most cases have FPG values below the putative threshold. If FPG is followed as a screening procedure no of pregnant women having GDM would be higher. For these reasons fasting glucose is not favoured by the WHO for diagnosing GDM.

Random plasma glucose:

Random plasma glucose measurement has been encouraged as a simple way to screen for abnormal glucose tolerance. A positive cut- off was taken as 6.4 mmol/l if the women were tested less than 2hr after a meal and 5.8 mmol /l if more than 2hr after a meal. Data on sensitivity & specificity of this method of screening was subsequently obtained which showed this to be a poor method of screening.

50 gm glucose challenge test:

50 gm of glucose is administered orally between 24-28 weeks at any time. Fasting is not required. Venous plasma glucose is measured 1 hr later.

If the cut off-point is set at 140 mg/dl (7.8 mmol/L) 80% of women with GDM will be detected. If this threshold for further testing is lowered to 130 mg/dl 90% of GDM cases will be detected.

If results of screening are positive, a 3 hour 100 gm OGTT should be carried out at 24-28 weeks for early identification of GDM. If the results of screening are normal, it should be repeated at 32-34 weeks especially in obese patients, elderly patients and women with risk factors for GDM.

A value of 200 mg/dl on screening is so likely to be associated with the diagnosis of GDM that the GTT need not be performed and treatment can be started. 50 gm GCT was found to be very sensitive in detection of gestational diabetes in high risk group. Coustan et al found that current ACOG recommendations result in sensitivity of 65%. Kini et al opined that 50 gm GCT should be repeated in 3rd trimester as it yields a large no of gestational diabetics. Due to the simplicity, acceptability, sensitivity and cost effectiveness of GCT,

it is the best method to detect gestational diabetes mellitus in high risk group.

75 gm glucose tolerance test.

WHO recommends performing 2 hour 75 g OGTT and diagnosing GDM with a threshold plasma glucose concentration greater than 140 mg/dl (7.8 mmol/L) at 2 hours similar to that of impaired glucose tolerance test in the non-pregnant. This method serves both as a one-step screening and diagnostic procedure and is easy to perform besides being economical.

Fructosamine assay

It is associated with glycemic control over the previous 1-3 weeks possibly making it a more appropriate marker for gestational diabetes. However its sensitivity is too low for it to be used as a screening method for GDM.

Glycosylated hemoglobin:

Proteins react spontaneously with glucose to form glycosylated derivatives. HbA_1c serves as a retrospective indicator of the average glucose concentration over the previous 8-10 wks. The HbA_1c level is more strongly correlated to preprandial than postprandial glucose concentrations. In the third trimester however

HbA_1c levels may only reflect mean glucose values over the previous 2 weeks, presumably because of increased rates of erythropoiesis. HbA_1c is expressed as a percentage of the normal hemoglobin and the normal range is approximately 4% to 6%.

The use of HbA_1c and glycosylated proteins has been extensively investigated as a simple one sample screening test with no preparation required. Unfortunately after initial enthusiastic reports, multiple studies have, perhaps not surprisingly, shown that the tests have poor specificity and sensitivity.

Diagnosis of GDM

The diagnostic test for GDM is the oral GTT. In North America, the American Diabetes Association and National Diabetes Data group recommend a 100 g oral glucose challenge as the diagnostic test and a randomly administered 50g oral glucose challenge as the screening test.

100gm of oral glucose is administered in the morning after an overnight fast of 8-14 hours duration after 3 days of carbohydrate rich diet(i.e. unrestricted diet $\geq$150 gm of carbohydrate per day) and unrestricted physical activity.

The subject should remain seated and should not smoke throughout the test. The fasting venous blood glucose samples are taken at 1,2 & 3 hours.

Criteria for the diagnosis of GDM with 100gm oral glucose (venous plasma mg/dl)				
Time	O'Sullivan	Normal diabetes data group (1979)	Carpenter and coustan (1982)	WHO
Fasting	90	105	95	≥140
1 hr	165	190	180	
2 hrs	145	165	155	≥200
3 hrs	125	145	140	

The diagnosis of GDM is established if any 2 or more values exceed the upper limit of normal.

If only one value is abnormal the patient cannot be diagnosed as having GDM although she is at high risk. They have insulin resistance comparable to patients with GDM and are more likely to deliver macrosomic infants.

Criteria for diagnosis of impaired glucose tolerance and diabetes with 75gm oral glucose (ADA) plasma (mg/dl)			
Time	Normal tolerance	Impaired glucose tolerance	Diabetes Mellitus
Fasting 2hrs pp	<110 <140	≥110 - <126 ≥140 - <200	≥126 ≥200

Impaired glucose tolerance is diagnosed if fasting level is between 110 - 125 mg/dl or 2 hours postprandial is between 140 – 199 mg/dl.

Diabetes is diagnosed if,

Fasting is ≥ 126 mg/dl

2 hour PP is ≥ 200 mg/dl in plasma and 170 mg/dl in whole blood.

Venous whole blood values are 15 percent less than the plasma values.

Women with GDM are undoubtedly at increased risk for adverse obstetric and perinatal outcomes. Good maternal and fetal outcomes result from early and meticulous prenatal and intranatal care. Thus all pregnant women should be screened for GDM at least

once during pregnancy irrespective of the presence or absence of risk factors and all detected GDM, should be closely monitored for strict glycaemic control throughout pregnancy for optimal neonatal outcome.

Hence based on the above data this study was designed to screen pregnant women universally for GDM. 50g GCT and random Blood glucose was performed on 200 pregnant women belonging to 24 to 28 weeks gestation. For all screen positive cases, 3 hour OGTT with 100g glucose was performed.

MATERIALS AND METHODS

SELECTION OF CASES:

1. 200 pregnant women in their 24-28 weeks of gestation were selected irrespective of parity, age and risk factors.
2. All the 200 pregnant women had random blood glucose and GCT done.

INCLUSION CRITERIA

1. All women who were selected had clinical examination and when the gestation was between 24-28 weeks were included in this study.
2. Women who were not sure of their last menstrual period and whose clinical examination was inappropriate ha an ultrasound examination and when the period of gestation was between 24-28 weeks they were included.

EXCLUSION CRITERIA

1. Women who were not within 24-28 weeks of gestation were excluded from the study.
2. Women who were already pre-gestational diabetes and proved gestational diabetics in their current conception were excluded.

PLACE OF STUDY

Govt Rajaji Hospital, Madurai Medical College, Madurai.

YEAR OF STUDY

2009

NATURE OF STUDY

Prospective study

METHOD

Pregnant women who were selected with the above criteria had their random blood glucose estimated and were given 50g glucose drink, without prior patient preparation. Glucose given-Dextrose monohydrate D-glucose. One hour later 2.5ml of venous blood was drawn in an oxalate and fluoride test tube and immediately handed over to our hospital laboratory. Following the glucose drink patients were prohibited from further eating or drinking except water.

ESTIMATION OF BLOOD GLUCOSE

ORTHOTOLUIDINE METHOD

0.1ml plasma

5 ml of orthotoluidine reagent. Boil for 8 minutes and take reading at 650nm in a calorimeter. Glucose reacts with orthotoluidine in acid medium to give green color. The blood sugar

levels obtained by this method were also confirmed by enzyme method.

ENZYME METHOD

Glucose is oxidized by glucose oxidase into gluconic acid and H_2O_2. H_2O_2 in the presence of peroxidase (POD) oxidizes the chromogen 4 aminophenozone/phenolic compound to a red colored compound. The intensity of the red color produced is proportional to the glucose concentration and is measured at 505nm (490-530nm). The final color is stable for two hours.

$$\text{Glucose} + O_2 \xrightarrow{\text{glucose oxidase}} \text{gluconic acid} + H_2O_2$$

$$H_2O_2 \text{ ===➔ } H_2O+O$$

$$\text{Chromogen (Colour less)} \xrightarrow{\text{Nascent oxygen}} \text{coloured product}$$

Venous blood is collected in a fluoride containing test tube. Plasma is preferred. Sodium fluoride is added to prevent glycolysis.

All women who had blood sugar value of $\geq$ 130 mg/dl or 140 mg/dl were considered screen positive and were subjected to 3 hours 100 gms OGTT within a week of screening.

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2008).

Information Package (EPI 2008)

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's test for qualitative variables. A'p' value less than 0.05 is taken to denote significant relationship.

Sensitivity, specificity, accuracy, positive predictive value and negative predictive values were calculated using the following formulae and taking GTT as the Gold standard.

$$\text{Sensitivity} = \frac{\text{True positive}}{\text{True positive + False negative}} \times 100$$

$$\text{Specificity} = \frac{\text{True negative}}{\text{False positive + True negative}} \times 100$$

$$\text{Accuracy} = \frac{\text{True Positive + True Negative}}{\text{Total cases}}$$

$$\text{Positive predictive value} = \frac{\text{True positive}}{\text{True positive + False positive}} \times 100$$

$$\text{Negative predictive value} = \frac{\text{True negative}}{\text{True negative + False negative}} \times 100$$

OBSERVATION AND RESULTS

1. AGE DISTRIBUTION

Age Group (in years)	Cases	
	No	%
17-20	33	16.5
21-25	105	52.5
26-30	52	26
31-35	10	5

Age of pts ranged from 18 - 35 years. In our study 5% of the pts were more than 30 years among which one was a GDM Patient. No GDM patient had age less than 21 years. Mean age of the patient under our study was 24.1 years.

2. EDUCATIONAL STATUS

Literacy	Non GDM	%	GDM	%
Illiterate	61	30.5	0	0
Primary education	55	27.5	1	33.33
High School education	73	36.5	2	66.66
College education	8	4	0	0

Majority of non GDM pts had high school education.

3. SOCIOECONOMIC STATUS

Socioeconomic status	No of cases in non GDM patients	%	No of cases in patients with GDM	%
Group I				
Group II				
Group III	17	8.5	2	66.66
Group IV	87	43.5	1	33.33
Group V	93	46.5		

Majority of non GDM patients belonged to Group V

4. PARITY

Parity	Cases	
	No	%
Primi	80	40
G2	89	44.5
G3	20	10
G4	10	5
G5	1	0.5
Total	200	100

In our study majority of patients were primi and second gravida.

5. BODY MASS INDEX.

BMI	Cases	
	No	%
<18.5	43	22%
18.5-24.9	127	63%
25 - 29.9 (overweight)	28	14%
30 and above (obese)	102	1%

In our study mean BMI of the patients was 21.5. All 3 GDM patients had BMI between 25 to 29.9 and belonged to over weight category.

6. FAMILY H/O DIABETES

Family History	No of cases in 200 pregnant women	%	No of cases in pts with GDM	%
Father DM	13	6.6	1	33.33
Mother DM	9	4.6	1	33.33
Both parents DM	2	1		
No of patients with Family h/o DM	24	12.18	2	66.66

Among patients with GDM, 1 had family h/o mother with DM, 1 had family h/o father with DM. In our study family h/o Diabetes mellitus was present in 66% of patients with GDM.

7. RISK FACTORS IN PRESENT PREGNANCY

Risk of Factors	No of cases in non GDM pts	%	No of cases in pts with GDM	%
Overweight, obese	30	15%	3	100
H/O Recurrent UTI	3	1.6%	1	33.33
H/O moniliasis	2	1.2%	1	33.33
PIH	16	8	2	66.66
Hydramnios	7	3.5	2	66.66
Glycosuria	20	10	3	100
Congenital malformation	5	2.5	-	

Among GDM patients, all 3 was overweight 2 had PIH, 2 had hydramnios, all 3 had glycosuria, 1 had h/o Recurrent UTI, 1 had h/o moniliasis. The same patient had more than one risk factor.

8. RISK FACTORS IN PAST PREGNANCY.

Risk Factors	No of cases in non GDM pts	%	No of cases in pts with GDM	%
H/O spontaneous abortion	25	12.6	1	33.33
H/O delivery of a baby with macrosomia	8	4		
H/O sudden IUD	3	1.5	1	33.33
H/O preterm delivery	-	-		
H/O still birth	1	0.5		
H/O instrumental delivery	2	1		
H/O unexplained neonatal death	3	1.5		
H/O previous GDM or IGT	-	-		
H/O previous PIH	-	-	1	33.33
H/O previous Babies with congenital anomalies	1	0.5		

Among GDM patients one had h/o spontaneous abortion, h/o sudden IUD and h/o PIH in her previous pregnancy.

9. DISTRIBUTION OF BLOOD GLUCOSE VALUES IN GCT AND RANDOM BLOOD GLUCOSE

Blood glucose levels (Range)	GCT		RBG	
	No	%	No	%
61-70	13	6.5	12	6
71-80	10	5	19	9.5
81-90	27	13.5	40	20
91 - 100	34	17	45	22.5
101-110	51	25.5	35	17.5
111-120	22	11	25	12.5
121-130	10	5	11	5.5
131-140	9	4.5	6	3
141-150	11	5.5	6	3
151-160	4	2	1	0.5
161-170	5	2.5	-	-
171-180	2	1	-	-
181-190	2	1	-	-
Range	60-186		60-155	
Mean	106.9		98.7	
SD	24.9		19.3	

P = 0.00007 significant

10. RESULTS OF OGTT AT 24-28 WKS

S.No	GCT	Random Blood glucose	GTT				OGTT
			105	190	165	145	
			F	1 hr	2 hr	3 hr	
1	144	138	85	140	126	110	
2	165	150	60	117	96	88	
3	137	128	93	158	122	110	
4	145	138	60	114	126	101	
5	152	140	62	123	102	68	
6	176	150	85	175	128	106	
7	146	112	91	175	140	105	
8	132	120	82	155	132	104	
9	167	146	88	146	103	96	
10	173	155	96	179	111	100	
11	186	140	62	173	89	80	
12	156	130	75	119	108	100	
13	142	136	88	162	113	105	
14	163	146	80	162	126	100	
15	139	116	82	140	130	105	
16	146	113	70	98	96	90	
17	146	117	72	120	78	70	
18	168	142	72	156	122	90	
19	**170**	**142**	**95**	**195**	**170**	**140**	**+ve**
20	143	122	87	166	132	100	
21	141	121	81	162	136	98	
22	181	135	91	166	142	98	
23	140	118	89	112	83	72	
24	**138**	**120**	**119**	**169**	**186**	**132**	**+ve**
25	136	122	88	170	135	99	
26	143	124	90	165	146	102	
27	137	124	82	140	116	96	
28	154	118	82	130	126	106	
29	133	120	72	170	96	82	
30	**150**	**122**	**145**	**205**	**170**	**115**	**+ve**
31	152	123	93	170	122	102	
32	136	122	80	120	76	70	
33	150	120	84	162	115	102	

11 - DETECTION OF GDM BY VARIOUS TESTS

Test	Cut off value of Blood glucose	Positive		Negative	
		No.	%	No.	%
GCT	140	24	12	176	88
GCT	130	33	16.5	167	83.5
RBG	126	16	8	184	92
GTT		3	1.5	197	98.5

12 - MANAGEMENT OF DETECTED GDM CASES

Management	Cases	
	No.	%
Diet Control	1	33.33
Insulin	2	66.7

Tight glycemic control could be achieved by diet control alone in one patient. Other 2 patients required insulin whose requirement varied from 15-25 units in 24 hrs.

Patients were followed with

1. Meticulous blood glucose control such that in the fasting, blood glucose was <95 mg/dl and 2 hr postprandial blood glucose <120mg/dl .
2. Daily fetal movement count (kick-chart)
3. Serial ultrasonogram to determine estimated fetal weight, fetal growth profile, congenital anomalies and amniotic fluid index.

DETAILS OF OGTT +VE WOMEN.

1. Primi, 31 years of Grade III socio economic status having high school education married 6 yrs back (Non consanguinous marriage with no family ho diabetes. Her height was 148 cm and she weighed 62kg. BMI was 28.30.

GCT	RBG	OGTT			
		F	1hr	2hr	3hr
170	142	95	195	170	140
+ve	+ve	+ve			

She had PIH in the present pregnancy for which she was treated with alphamethyldopa 250mg 2tds. She was managed with diet control alone and she was delivered by LSCS after 37 weeks. Indication being mobile breech with PROM with long period of infertility. It was an alive preterm female baby weighing 2kg with apgar score 1min 6/10. Her post operative period was uneventful. She came for follow up 6 weeks after delivery and it was found that her blood glucose values returned to normal.

2. G_2 P_1L_1, 25 yrs of age of grade III socioeconomic status having high school education married 3yrs back consanguinous marriage

with family h/o diabetes in the mother. Her height was 157 cm and she weighed 65kg. BMI 26.37. Her previous pregnancy was full term normal delivery.

GCT	RBG	OGTT			
		F	1 hr	2 hr	3 hr
150	122	145	205	170	115
+ve	-ve	+ve			

She had h/o recurrent UTI and moniliasis for which treatment given. She was managed with insulin and had spontaneous onset of labour after 37wks. An alive term male baby was delivered by outlet forceps with episiotomy weighing 3.5kg with 1min Apgar score 8/10. Baby had hypoglycemia for which treatment was given. Baby recovered well. Her postpartum period was uneventful. She came for follow up 6 wks after delivery and she was found to have normal glucose values.

3. $G_4P_1L_oA_2$ belonging to grade IV socioeconomic status had primary education married 5 yrs back (consanguinous marriage) with family h/o diabetes in the father. She had 2 spontaneous abortion both certified at 2 and 3 months respectively and in the next

pregnancy she had sudden IUD at 8 months of gestation. She had h/o PIH in that pregnancy for which she was treated. Her height was 156 cm and weighed 62kg BMI 25.47.

GCT	RBG	OGTT			
		F	1hr	2hr	3hr
138	120	119	169	186	132
-ve	-ve	+ve			

She had PIH in the present pregnancy. She was managed with labetalol 100 mg bd and started on insulin and delivered by LSCS at 34wks of gestation as the doppler study showed early diastolic notch in uterine artery and abnormal high resistance flow in umbilical artery. Indication was BOH with GDM with severe PIH. It was an alive preterm male baby weighing 1.8kg with 1min Apgar 6/10. On admission to preterm ward baby had respiratory distress syndrome and had jaundice for which treatment given. Baby was discharged after 2 wks. Pt had postoperative wound infection which healed with antibiotics. She reported for follow up 6 weeks after delivery and she was found to have impaired glucose tolerance. Pt was advised lifestyle modification and regular follow up.

Postnatal follow up using 2 hr 75g Oral glucose tolerance test.

Post natal	No of cases in patients with GDM	%
Reversion back to normal	2	66.67
Impaired glucose tolerance	1	33.33

Six weeks, after delivery, 2 reverted back to normal and 1 had impaired glucose tolerance.

DISCUSSION

A prospective study was conducted in Govt. Rajaji hospital during the year 2009. For this study, 200 randomly selected pregnant women in their 24 to 28 weeks of gestation were included. 50g Glucose challenge Test and Random Blood Glucose was done without prior patient preparation in all 200 randomly selected pregnant women. The study was designed to find out whether 50g Glucose Challenge Test (or) Random Blood Glucose is better in a set up like our hospital to detect gestational diabetes.

PREVALENCE:

The prevalence of abnormal Glucose Tolerance is highly dependent upon ethnicity (Hadden 1985[27] and Beischer et al 1991[4]) Stephen et al in 1981 found in his study that the incidence of gestational diabetes lies between 1 to 5%.

Ranchod et al[57] found the prevalence of gestational diabetes in the Indian subcontinent 1.6% by applying WHO GTT Diagnostic criteria.

Ramachandran A[56] carried out screening for gestational diabetes in 950 patients in southern India showed the prevalence of

GDM to be 0.56%. Initially screening test was done with the 50g glucose load and values more than or equal to 140mg/dl were subjected to 3hr oral GTT. According to Mudaliar, the incidence of diabetes varies from 0.3to0.7%. In our study of 200 unselected pregnant women, when screened between 24-28 weeks of gestation, the prevalence of GDM was found to be 1.5%.

At 24-28 weeks results of the glucose challenge test is as follows:

Gabbe et al[25] in 1991 concludes that the Glucose Challenge Test is the best Screening Test for abnormal Glucose Tolerance. The most recent ACOG technical bulletin suggests that 15% of all women in a given population would be expected to have abnormal GCT.

According to Williams when 140 mg/dl is used as threshold, 14-18% will have positive tests, when 130mg/dl is used as threshold, 20-25% will have positive tests. In our study out of 200 randomly selected pregnant women when threshold used was 140mg/dl(7.8mmol/L) 24 women i.e. 12% were screen positive.

If the cut-off value was reduced to 130mg/dl(7.2mmol/L), 33 women (16.5%) were screen +ve. Therefore in our study among 200

pregnant women between 24 and 28 weeks of gestation, GCT was +ve in 12% of cases.

Originally an overall sensitivity of 79% and a specificity of 83% was reported by O'Sullivan et al[53]. In our study of 200 randomly selected pregnant women, O'Sullivan's test had a sensitivity of 67% and a specificity of 89%.

Coustan et al[11] 1993 conducted a study, in which he maximized sensitivity by using a 130mg/dl cut-off and he was able to increase specificity at the sacrifice of some sensitivity by using a 140mg/dl cut-off. In our study on 200 randomly selected pregnant women, when 140mg/dl was used as cut-off sensitivity was 67% and sensitivity increased to nearly 100% when 130mg/dl was used as cut-off.

At 24-28 weeks results of Random Blood Glucose test is as follows:

Mathai et al, Thomas. T.J. et al[46] studied in 121 pregnant women with no risk factors using casual plasma glucose estimation and found that it had a sensitivity of 63% and specificity was 66%. Hadden used random plasma glucose above 120mg/dl irrespective of meals as an indication for full testing.

In a study by Jardine Brown et al[28], a recent specialist UK workgroup Report on Diabetes in Pregnancy has suggested that women from low-risk populations should be offered a random blood glucose estimation at 28 weeks gestation together with further random blood glucose measurements whenever glycosuria is observed. If these are >6mmol/L (>108mg/dl in the fasting state or 2hrs after food, or >7mmol/L (126mg/dl) within 2hrs of food, then a GTT should be performed. Originally a sensitivity of 50% and a specificity of 90% was reported. In our study random blood glucose had a sensitivity of 33% and a specificity of 92%.

Using these criteria, random blood glucose estimation was done. Since almost all women attending our Antenatal O.P came within 2 hrs of food, 126mg/dl was used as the cut-off value for random blood glucose in our study. Out of 200 pregnant women screened, 16 women (8%) were screen +ve. Therefore in our study among 200 pregnant women between 24 and 28 weeks of gestation, Random blood glucose was +ve in 8% of cases, OGTT confrmed the diagnosis in 6.25% of these positive tests.

Oral Glucose tolerance test

OGTT was done on all women who were screen positive the results were interpreted using NDDG criteria.

1 patient had GCT positive, random blood glucose positive and OGTT positive, second pt had GCT positive, random blood glucose negative, but OGTT positive. Third pt had GCT negative, random blood glucose negative, but OGTT positive.

According to the 1986 ACOG technical bulletin, 15% of those undergoing 3hr OGTT could be anticipated to have an abnormal result. In our study 10% of those undergoing 3hr OGTT had an abnormal result.

In the Toronto-tri hospital study, women who had “borderline” GDM (met carpenter and coustan’s criteria but not NDDG criteria) and had one abnormal value using the NDDG criteria, had twice the rate of macrosomia as women who had normal glucose testing (28% versus 13%).

So 3hr OGTT was done on all screen positive women and the results were interpreted using NDDG (National Diabetes Data Group) criteria. Out of this 3 women were found to have gestational

diabetes, among them 2 had family history of diabetes and one had h/o sudden IUD

UNIVERSAL SCREENING VS SELECTIVE SCREENING

According to American college of obstetrics and Gynaecology all pregnant patients should be screened for gestational diabetes. But according to American diabetes association, screening should be done for women with risk factors alone because of cost-benefit considerations.

In a study by Danilenko Dixon DR, van winter J.T. nelson Riet al[17] applying ADA critieria, 3% of gestational diabetics would have gone undiagnosed.

In a study by coustan et al[11] on 6214 women if screening is done based on risk factors alone, 35% of all cases of a GDM would be missed.

Howard et al recommended screening with 50g oral glucose load followed by a 3hr GTT for women with risk factors alone and found that 30-50% of women with gestational diabetes were overlooked.

Dradi, Maraldi.C Confirmed that O'Sullivan's GCT at 24-28 weeks of gestation in all pregnant women is a must since GDM is often pauci or asymptomatic which is associated with an increase of

maternal fetal and neonatal morbidity plus an increased risk of later overt diabetes mellitus in the mother.

Baxi L, Singh. S, Sacks, D[2] concluded that plasma glucose obtained 1hr after 50g oral glucose challenge is the best GDM Screening Test.

If only women with high risk factors are screened approximately 35% of GDM patients will be missed. So screening should be universal. Universal screening for GDM is justified by morbidity reduction, protocols simplicity and ease.

MATERNAL OUTCOME

Age:

Moses et al[55] in his study showed that age more than 30yrs is present in 8.5% of GDM Patients and age younger than 21 years is present only in 0.7% of GDM Patients.

In our study age more than 30yrs was present in 33.33% of GDM Patients and age younger than 21yrs was not present in any GDM patient.

Parity

Pyke DA et al[55]found that the incidence of diabetes increased with increasing parity. But according to recent reports parity has no

predictive value for diabetes and is unlikely to play an etiological role.

In our study the incidence of GDM was slightly higher in multi gravidas.

BMI

Serirat et al 1992 in a study found that Obesity was present in 26.5% of patients with GDM. Landon and colleagues (1994) and zhang and co-workers (1995) found that the risk of gestational diabetes was increased in women with truncal Obesity. (Williams Obstetrics)

In our study pts with GDM were in the Overweight category BMI 25-29.9

Family History:

Serirat et al[68] in 1992 has shown that family H/O diabetes is present in 23.1% of Patients with abnormal glucose tolerance.

Moses et al[49] in 1995 has shown that family H/O diabetes is present in 11.6% of patients with GDM.

In our study family H/O diabetes Mellitus was present in 66.66% of Patients with GDM.

IN ANTENATAL PERIOD:

PIH:

Suhonen and Terano [64] in 1993 reported the incidence of PIH and pre-eclampsia to be 2 times more common among GDM patients than controls (19.8%Vs 10%).

Siddigi T, Rossen.B, Mimouri F et al[61] in their study found incidence of PIH in GDM to be approximately 15% compared to 7.7% in controls.

In our study PIH was found in 66.66% of pregnant diabetic patients.

HYDRAMNIOS:

Biggio et al in 1999 reported that hydramnios occurs in 20 % of diabetic pregnancies. Rosenn et al[74] found incidence of hydramnios to be 26.4% in their study.

In our study hydramnios was found in 33.33% of GDM Patients.

MANAGEMENT:

Langer.O, Rodri Guez. D.A, Xenakis E.M. et al[43] 30-60% of patients with GDM were treated with insulin. In our study 66.66% of GDM Patients were treated with Insulin.

IN THE INTRA-PARTUM PERIOD

Time of Delivery:

In a study by Goldman et al, pre-term delivery occurred in 9.4% of patients with GDM compared to 6.9% in controls. Preterm delivery is more common in women with pre-gestational diabetes than in women with GDM.

In our study 66.66% of patients with GDM had Preterm Delivery.

Mode of delivery

Goldman et al[24] in the study found caesarian section rates to be as high as 35% compared to 22% in normal controls.

Hawthorne G.Robson et al[32] in her study found caesarian section rates as high as 60-65%. In our study caesarian secion rates was almost 67%.

In the Post-Partum Period

Jacobson and cousins et al[27] in a study found post-caesarian infection in patients with GDM to be 12.4% compared to 5.9% in controls.

Maternal Mortality

Maternal deaths have become rare in women with diabetes, although as emphasized by cousins (1987), mortality is increased 10-fold, most often as a result of ketoacidosis, underlying Hypertension, preeclampsia, pyelonephritis and patients with coronary artery disease (class H).

In our study there was no maternal mortality.

FETAL OUTCOME

Birth Weight:

Spellacy, W.N.Mills and winger.A[65] found that macrosomia is present in 50% of pregnancies in patients with GDM. But lowering of birth weight by treatment has been shown in many studies including those of O'Sullivan's et al.

In our study one patient had delivered a baby weighing 3.5kg

Neonatal Complications:

Hypoglycemia:

Gabbe et al[25]observed that 99 babies out of 257 infants of diabetic mothers(39%) became hypoglycemic after delivery. According to James High risk pregnancy, the frequency of hypoglycemia is 18-49%.

In our study Hypoglycemia was present in 33.33% of babies born to patients with GDM.

Hypocalcaemia:

Marshall R.E in his study found the incidence of Hypocalcaemia in infants of diabetic mothers to be 50% . Kitzmiller J. Cloherty, J et al[39] found that the incidence of hypocalcaemia in infants of diabetic mothers to be 5-22% (Michael de Swiet).

In our study, hypocalcaemia was not found in any of the babies.

Hyperbilirubinemia:

Beard and lowy et al[3] found that the incidence of jaundice in infants of diabetic mothers to be 20% .

In our study Hyperbilirubinemia was found 33.33% of babies born to patients with GDM.

Respiratory Distress Syndrome:

Several studies agree that in well controlled diabetic patients delivered at term, the risk of RDS is no higher than that observed in the general population (10-15%).

In our study RDS was present in 33.33% of babies born to patients with GDM.

Perinatal Mortality:

All the 3 women with GDM, had good fetal outcome. Even the babies who had complications recovered well and there was no perinatal mortality.

STATISTICAL DATA

Validity of Glucose Challenge test

Statistical attributes of Glucose challenge test as a screening test for the detection of Gestational diabetes were analyzed and found as follows:

Cut off ≥ 140 mg / dl

GCT (≥140mg/d1)	OGTT			
	Positive		Negative	
	No.	%	No.	%
Positive (24)	2	8.3	22	91.7
Negative (176)	1	0.6	175	99.4

True positive = 2

False positive = 22

True Negative = 175

False negative = 1

Sensitivity = 67%

Specificity = 89%

Accuracy = 89%

Positive predictive value = 8%

Negative predictive value = 99%

P = 0.0385 significant.

Mean Blood glucose values 106.9mg

Standard deviation 24.9.

Cut off ≥ 130 mg / dl

GCT (≥130mg/dl)	OGTT			
	Positive		Negative	
	No.	%	No.	%
Positive (33)	3	9.1	30	90.9
Negative (167)	-	-	167	100

True positive 3

False positive 30

True Negative 167

False negative Nil

Sensitivity 100%

Specificity 85%

Accuracy 85%

Positive predictive value 9%

Negative predictive value 100%

P = 0.0041 significant.

prevalence 1.5%

INFERENCE:

Therefore reducing the threshold of GCT to ≥130mg/dl resulted in increase in sensitivity thereby yielding a higher diagnostic result.

Validity of Random Blood Glucose test

Statistical attributes of Random Blood Glucose test as a screening test for the detection of Gestational diabetes were analyzed and found as follows:

RBG (≥126mg/dl)	OGTT			
	Positive		Negative	
	No.	%	No.	%
Positive (16)	1	6.3	15	93.8
Negative (184)	2	1.1	182	98.9

True positive 1

False positive 15

True Negative 182

False negative 2

Sensitivity 33%

Specificity 92%

Accuracy 92%

Positive predictive value 6%

Negative predictive value 99%

P = 0.2223 Not significant.

Mean Blood glucose values 98.7

Standard deviation 19.3.

Screening test	Sensitivity %	Specificity %	PPV %	NPV %
GCT(≤140mg/dl)	67	89	8	99
GCT(≤130mg/dl)	100	85	9	100
RBG(≤126mg/dl)	33	92	6	99

INFERENCE:

GCT had a sensitivity of 67% and hence is for superior to Random blood glucose estimation.

GDM and family history of Diabetes

Family History of DM	OGTT			
	Positive		Negative	
	No.	%	No.	%
Present (26)	2	7.7	24	92.3
Absent (174)	1	0.6	173	99.4

X^2 =3.69

P =0.045(Significant)

GDM and Quantitative variables

Variable	Value for cases				'p'
	GDM cases		Non GDM cases		
	Mean	SD	Mean	SD	
Age	27.3	4.0	24.1	3.3	0.1443(Not significant)
Weight	63	1.7	49.1	8.5	0.0098(Significant)
BMI	26.7	1.4	21.4	3.6	0.0159(Significant)
a)GCT b) RBG	152.7 128	16.2 12.2	106.2 98.2	24.3 19.0	0.0104(Significant) 0.0139(Significant)

SUMMARY

1. In our study of 200 unselected pregnant women, prevalence of gestational diabetes was found to be 1.5%.
2. In detecting gestational diabetes in our population, the 50g Glucose Challenge Test had a sensitivity of 67% and specificity of 89% whereas Random Blood Glucose test had a sensitivity of 33% and a specificity of 92% . Hence it was found that Glucose Challenge Test was far superior to Random Blood Glucose estimation.
3. If the threshold in GCT was taken as 140 mg/dl, sensitivity was 67% whereas if the threshold in GCT was taken as 130mg/dl, the sensitivity was increased to nearly 100%.
4. Both Glucose challenge Test and Random Blood Glucose test do not require any prior patient preparation.
5. Oral Glucose Tolerance Test is the Gold Standard Diagnostic test for the diagnosis of gestational diabetes.
6. Universal screening for gestational diabetes has to be done since selective screening of women with high risk factors

alone is likely to miss more than 1/3rd of cases with gestational diabetes

7. In a population like ours, universal screening with 50g Glucose Challenge Test should be performed on all pregnant women between 24 and 28 weeks of gestation.

Hence early detection of gestational diabetes and effective management to maintain optimal blood glucose levels will drastically reduce maternal morbidity due to gestational diabetes and will bring about a definite reduction in perinatal mortality rate.

CONCLUSION

Universal screening for GDM has to be done with Glucose Challenge test to detect Gestational diabetes early irrespective of the presence or absence of risk factors.

All detected GDM patients have to be closely monitored for strict glycemic control throughout pregnancy for optimal maternal and neonatal outcome.

PROFORMA

Name of the patient : Date :

Husband's name : Age :

OP No. :

Address :

Socioeconomic Status :

Educational Qualification:

Obstetric code

LMP

EDD

Marital Status

Married since how many years.

Family history of DM
H/o preexisting Diabetes

H/o Risk factors in present pregnancy
- h/o recurrent UTI
- h/o moniliasis
- h/o PIH
- h/o Hydrammios
- h/o Glycosuria
- h/o Congenital malformations

H/o Risk factors in past pregnancy
- h/o spontaneous abortion
- h/o preterm delivery
- h/o Still birth
- h/o sudden neonatal death
- h/o difficult forceps
- h/o fetal macrosomia
- h/o previous PIH
- h/o previous congenital anomalies.

CLINICAL EXAMINATION

- Height
- Weight
- Gestational age

LABORATORY RESULT

- Blood glucose level following GCT
- Random Blood glucose
- OGTT values

INTERPRETATION.

ABBREVIATIONS

GCT	Glucose Challenge test
RBG	Random Blood glucose
OGTT	Oral Glucose Tolerance Test
FPG	Fasting Plasma Glucose
GDM	Gestational Diabetes Mellitus
PIH	Pregnancy induced hypertension
BMI	Body mass Index
BOH	Bad obstetric history
LSCS	Lower segment caesarean section
UTI	Urinary tract infection
LMP	Last menstrual period
EDD	Expected date of delivery

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S.No	Name	OP No	Obstetric Code	Age	Risk Factors	Weight	Height	BMI	GCT	RBG	OGTT			
											F	1hr	2hr	3hr
1	Pandisweeri	15459	Primi	23	Father Diabetic	49	150	19.6	90	86				
2	Priya	9369	G3P1L1A1	23	h/o Abortion	45	142	22.3	96	90				
3	Sikandar Maa	17788	G2P1L1	26	-	39	153	16.7	86	100				
4	Sharmila	19281	G2P1L1	31	Father Diabetic	43	158	17.2	84	72				
5	Rani	9486	G2P1L1	24	Mother Diabetic	38	152	16.4	78	84				
6	MANONMANI	15622	G2P1L1	28	Father Diabetic	41	141	20.6	80	93				
7	UMADEVI	14868	Primi	21	-	43	150	19.1	68	74				
8	PANDISELVI	15079	G3P1L1A1	25	h/o Abortion	50	153	21.4	98	90				
9	MUTHUMARI	22559	G3P2L1	29	h/o Macrosomia, h/o Sudden IUD	60	154	25.3	90	102				
10	GEETHA	9466	G2P1L1	25	-	60	147	27.8	100	94				
11	DEVI	20453	Primi	25	-	38	140	19.4	115	120				
12	PARVATHY	11009	G2P1L1	24	-	52	149	23.4	61	80				
13	SHALINI DEVI	28223	G3A2	26	h/o Abortion	51	142	25.3	115	102				
14	LAKSHMI	28237	G3P2L1	27	h/o neonatal death,h/o recurrent UTI	43	146	20.2	126	130				
15	BOTHAI AMMAL	28225	G2P1L1	25	PIH	56	155	23.3	99	94				
16	MAHESWARI	28217	Primi	24	-	62	145	29.5	99	89				
17	MUTHULAKSHMI	28216	Primi	20	-	42	143	20.5	87	82				
18	ESWARI	28233	G2P1L1	26	Father Diabetic, PIH	40	145	19.0	101	96				
19	MARAGATHAM	28437	G2P1L1	22	-	51	148	23.3	83	86				
20	PANDISELVI	28438	G2P1L1	21		60	143	29.3	66	72				
21	MEENAKSHI	28434	Primi	28	-	41	150	18.2	106	101				
22	YASMIN	28455	Primi	20	-	49	146	23.0	64	72				
23	SHAMBAGAM	18038	G5P4L1	27	h/o Abortion, h/o Sudden IUD, h/o Pre term Delivery, h/o recurrent UTI	40	145	19.0	144	138	85	140	126	110
24	KALEESWARI	28440	G2P1L0	22	h/o Stillbirth	50	148	22.8	68	72				
25	THASLEEMA	11058	Primi	20	Father Diabetic, PIH, Glycosuria	51	146	23.9	165	150	60	117	96	88
26	JAYA	11661	Primi	23	-	70	157	28.4	104	99				
27	DEVI	11662	Primi	20	-	43	157	17.4	68	62				
28	RATHIKA	11670	G2A1	27	h/o Abortion	66	158	26.4	76	74				

29	HEMALATHA	11669	G2P1L1	29	PIH	58	154	24.5	111	120				
30	ANEES PATHMA	11668	G2P1L1	21	Recurrent UTI	37	146	17.4	91	72				
31	TAMILARASI	6431	Primi	21	-	50	146	23.5	128	116				
32	KARISHMA	10517	G2P1L1	26	-	51	150	22.7	122	108				
33	GOKILA RANI	11664	G2P1L0	27	Father Diabetic, h/o Congenital Malformation Moniliasis	70	153	29.9	96	92				
34	RAKKU	11708	Primi	19	-	51	148	23.3	94	90				
35	SHANTI	11709	G2A1	24	h/o Abortion	52	148	23.7	100	86				
36	DEVI	11713	G2P1L1	30	-	49	148	22.4	107	98				
37	ANGALEESWARI	11727	Primi	20	-	41	149	18.5	137	128	93	158	122	110
38	SEKKI	11725	G2P1L1	22	-	49	152	21.2	119	112				
39	JAYAPRIYA	7868	Primi	18	-	51	149	23.0	101	96				
40	PODUMPONNU	9722	Primi	31	-	53	147	24.5	111	96				
41	KAMESWARI	11758	G2P1L1	20	Both Diabetic, h/o Macrosomia	56	160	21.9	105	115				
42	LAKSHMI	11314	G2P1L1	20	Moniliasis, Glycosuria	42	151	18.4	145	138	60	114	126	101
43	BACKIALAKSHMI	11760	G2P1L1	24	Glycosuria	52	153	22.2	152	140	62	123	102	68
44	RAJALAKSHMI	11769	G2P1L1	23	h/o Macrosomia	46	152	19.9	111	106				
45	MATHA	9525	Primi	25	-	41	152	17.7	107	101				
46	LATHA	19105	G2P2L1	24	PIH	56	150	24.9	68	70				
47	VASANTHA	8221	G2P1L1	23	-	46	156	18.9	176	150	85	175	128	106
48	KATHIJA BEEVI	20484	G2P1L1	23	Twins, h/o Macrosomia	53	155	22.1	80	82				
49	PANDEESWARI	8311	Primi	22	Heat Disease Compl	51	163	19.2	146	112	91	175	140	105
50	RAJALAKSHMI	20507	G2P2L1	26	-	38	152	16.4	113	106				
51	KAVITHA	29512	G2A1	20	h/o Abortion	60	158	24.0	103	99				
52	IRULI	9080	G2P1LO	27	h/o Sudden IUD	45	158	18.0	96	93				
53	MANIMEKALAI	11847	G2A1	20	h/o Abortion	40	148	18.3	132	120	82	155	132	104
54	KAMATCHI	11849	G3P2L2	20	-	40	150	17.8	97	88				
55	MURUKESWARI	11698	G2A1	22	Mother Diabetic, h/o Abortion	56	160	21.9	62	60				
56	PANCHAVARNAM	11900	Primi	22	-	48	146	22.5	111	102				
57	PONMAYIL	11952	G2P1L1	30	-	48	146	22.5	167	146	88	146	103	96
58	CHITRA	11016	G2P1L1	32	-	51	150	22.7	173	155	96	179	111	100
59	CHITRA	10551	Primi	21	-	39	147	18.0	101	96				

60	LAKSHMI	11905	G2A1	22	h/o Abortion	46	143	22.5	89	88				
61	PODUMPONNU	11947	G2P1L1	24	-	46	150	20.4	83	80				
62	DEVI	11963	G2P1L1	24	-	45	152	19.5	90	84				
63	MURUKESWARI	11967	Primi	24	-	44	152	19.0	82	70				
64	MEENA	12003	Primi	20	-	49	158	19.6	103	88				
65	NACHAMMAL	12008	G2P1L1	25	Glycosuria	52	154	21.9	129	120				
66	BRINDHA	84233	Primi	23	-	50	150	22.2	108	101				
67	ANGALEESWARI	12009	G2P1LO	27	Mother Diabetic, Glycosuria, h/o Sudden IUD	60	153	25.6	186	140	62	173	89	80
68	VEERAMMAL	12012	G2P1L1	29	Father Diabetic, Glycosuria	57	156	23.4	156	130	75	119	108	100
69	RAMJAN BEGAM	11930	Primi	21	Both Diabetic, Congential Malformation	44	154	18.6	108	96				
70	PONNU MUNIYAMMAL	11202	G2P1L1	22	Twins, Hydramnios	42	147	19.4	99	90				
71	KALAISELVI	12024	Primi	20	-	44	155	18.3	101	98				
72	UMA	12026	G2P1L1	24		47	153	20.1	76	70				
73	SIVAGAMI	12027	G2P1L1	24	PIH	40	144	19.3	118	102				
74	PANCHU	12036	G2P1L1	24		58	141	29.2	108	106				
75	RADHA	12040	G2P1L1	27		39	152	16.9	77	70				
76	SUBATHRA DEVI	7657	G2P1L1	22	-	48	148	21.9	89	82				
77	SAKTHI MAI	12050	Primi	26	-	43	156	17.7	112	107				
78	KALAIVANI	10459	Primi	23	-	43	152	18.6	123	118				
79	LINGESWARI	12100	G2P1L1	27	PIH, Glycosuria	57	152	24.7	142	136	88	162	113	105
80	MALATHI	12111	G3P2LO	32	Congential Malformation, h/o Pre term Delivery, h/o Stillbirth	56	156	23.0	93	90				
81	DURGADEVI	30085	Primi	22	Glycosuria	44	149	19.8	163	146	80	162	126	100
82	MALIGA	9618	Primi	30	-	44	159	17.4	123	118				
83	NOORSHANT NISHA	8134	G2P1L1	27	Glycosuria	44	161	17.0	139	116	82	140	130	105
84	VIJAYARANI	11165	G3P1L1A1	24	h/o Abortion, h/o Macrosomia	53	151	23.2	110	101				
85	RAJESWARI	12129	G4P3L1	21	Glycosuria, h/o Sudden IUD, h/o Stillbirth, h/o Outlet Forceps	66	150	29.3	146	113	70	98	96	90
86	MANIPRABHA	12130	G2P1L1	24	Glycosuria	57	143	27.9	146	117	72	120	78	70
87	MAHESWARI	5832	Primi	21	Glycosuria	46	142	22.8	168	142	72	156	122	90
88	KANIMOZHI	25914	Primi	23	Father Diabetic	46	157	18.7	86	80				
89	MEENA	10755	Primi	20	-	46	155	19.1	83	77				

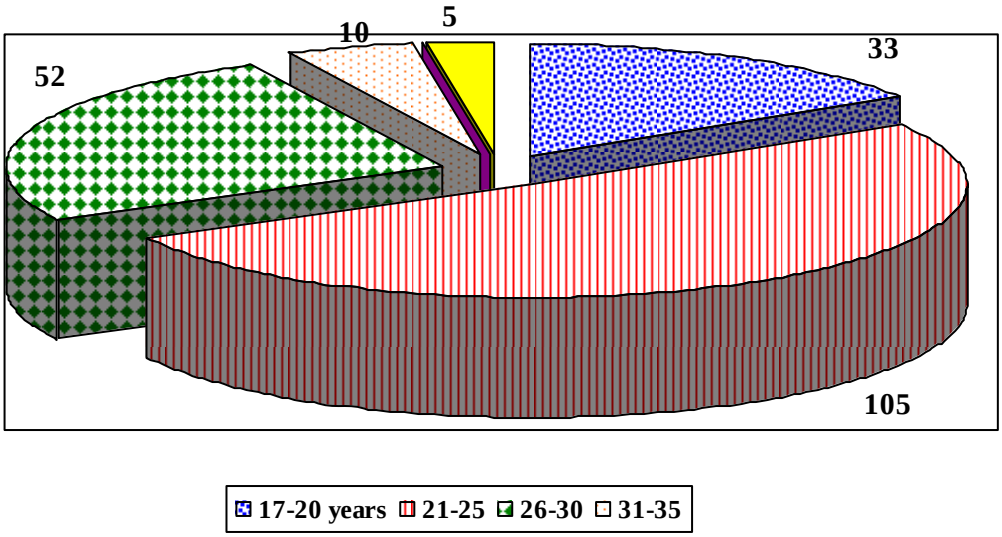
90	NAGESWARI	12138	Primi	23		50	150	22.2	104	87				
91	PRIYADHARSINI	12146	G2P1L1	25	-	59	155	24.6	128	120				
92	SASIKALA	10513	G2P1L1	27	h/o Outlet Forceps	40	151	17.5	60	66				
93	SHANTI	12144	G2P1L1	25	-	51	152	22.1	97	92				
94	SUNDHARI	29911	Primi	31	Glycosuria, Hydramnios, PIH	62	148	28.3	170	142	**95**	**195**	**170**	**140**
95	AMBIKA	12240	G2P1L1	25	-	56	157	22.7	106	102				
96	RUBINI	12244	Primi	18	-	47	148	21.5	89	82				
97	SATHYA	12250	Primi	19	Glycosuria	47	165	17.3	143	122	87	166	132	100
98	SURIYA	12253	Primi	19	-	61	157	24.7	101	97				
99	CHANDRA	10539	G3P3L2	31		46	150	20.4	98	90				
100	SUBERIA BANU	12291	Primi	20	-	57	153	24.3	141	121	81	162	136	98
101	RUKMANI	9283	Primi	23	-	48	165	17.6	94	92				
102	GANDHIMATHI	6583	Primi	30	-	54	148	24.7	181	135	91	166	142	98
103	VANITHA	8251	Primi	25	0	72	156	29.6	104	88				
104	VANITHA	12303	Primi	32	0	52	149	23.4	140	118	89	112	83	72
105	SUGANYA	11375	G3P1LO	20	h/o Abortion, h/o Congenital malformation	48	146	22.5	81	62				
106	MARUTHAYI	12348	Primi	24	0	40	152	17.3	82	68				
107	RAJESWARI	91636	G2P1L1	25	PIH	50	150	22.2	106	92				
108	RAMAPRIYA	5489	Primi	21	0	38	146	17.8	92	86				
109	SUBHASHINI	11621	Primi	20	0	56	152	24.2	88	80				
110	MARY PUSHPA RANI	42812	G4P1L0A2	28	Father Diabetic, PIH, Glycosuria, h/o Abortion, h/o Sudden IUD, h/o Previous PIH	62	156	25.5	138	120	**119**	**169**	**186**	**132**
111	MALARKODI	8839	G2P1L1	24	Mother Diabetic	46	146	21.6	107	101				
112	VEERAMMAL	11129	Primi	21	0	52	158	20.8	70	62				
113	SABIA BEGAM	9079	G2P1L1	24	0	48	148	21.9	109	101				
114	RAHMAT BEEVI	10185	Primi	21	0	46	152	19.9	136	122	88	170	135	99
115	MAHALAKSHMI	23193	G2P1L1	24	0	45	148	20.5	104	100				
116	DHANALAKSHMI	9367	G2P1L1	26	0	60	156	24.7	86	82				
117	SATHYA	12393	G2A1	23	Mother Diabetic,h/o Abortion	40	162	15.2	143	124	90	165	146	102
118	PARVATHY	12394	G2P1L1	21	0	48	150	21.3	137	124	82	140	116	96
119	CHINNAPILLAI	12397	G4P3L1	25	h/o Sudden IUD	40	150	17.8	154	118	82	130	126	106

120	KRISHNAVENI	8312	G2P1L1	24	Glycosuria	63	150	28.0	133	120	72	170	96	82
121	ANNALAKSHMI	41478	G4P1L1A2	23	h/o Abortion	68	154	28.7	125	116				
122	VANI	12293	G2P1L1	26	Father Diabetic	46	148	21.0	107	99				
123	VELUMANI	8988	G2A1	26	h/o Abortion	50	152	21.6	90	86				
124	PATHLA BANU	92168	Primi	19		51	155	21.2	108	98				
125	MALIGA	20116	Primi	28		59	142	29.3	96	90				
126	KAVITHA	11233	Primi	20	Mother Diabetic	70	160	27.3	89	82				
127	OYYAMMAL	12211	G4P2L2A1	35	h/o Abortion	60	160	23.4	89	82				
128	KALEESWARI	30772	Primi	20	PIH	41	142	20.3	93	84				
129	AMUDHA	12315	G2P1L1	26		77	161	29.7	97	86				
130	SHARMILA	10642	G2A1	24	h/o Abortion	67	160	26.2	104	92				
131	CHANDRA	9017	G3P2L2	27	h/o Macrosomia	56	152	24.2	83	80				
132	KAVITHA	11954	G3P2L2	23	h/o Macrosomia	40	158	16.0	103	92				
133	KAVITHA	11748	Primi	23		35	150	15.6	96	88				
134	PANDIAMMAL	9550	G2P1L1	23	h/o Macrosomia	50	151	21.9	105	100				
135	LAKSHMI KALA	11063	Primi	25	Father Diabetic	48	155	20.0	84	76				
136	MUTHU SARASWATHI	11464	Primi	23		63	153	26.9	103	97				
137	INDRA	30673	Primi	30		50	159	19.8	111	101				
138	MUTHULAKSHMI	1209	G3P1L1A1	30	h/o Abortion	50	161	19.3	106	102				
139	VIJAYA	8255	G4P2L1A1	23	h/o Abortion, h/o neonatal Death, PIH	50	155	20.8	98	92				
140	AYAMMAL	14922	G2P1L1	30		41	148	18.7	97	94				
141	SHANTI	3509	Primi	23	Father Diabetic	50	142	24.8	78	72				
142	SUMATHI	12126	Primi	23		50	155	20.8	104	96				
143	CHITRA	12140	Primi	25		50	152	21.6	108	98				
144	MAHALAKSHMI	12385	G3P1L1A1	29	h/o Abortion	46	148	21.0	96	100				
145	MEENAKSHI	12331	Primi	23	Hydramnios	40	144	19.3	106	108				
146	SHANTI	5799	G4P2LOA1	26	Hydramnios, h/o Abortion, h/o Stillbirth	45	150	20.0	106	102				
147	POORNIMA	10643	G2P1L1	23	Mother Diabetic, Recurrent UTI, Moliasis, Glycosuria	65	157	26.4	150	122	**145**	**205**	**170**	**115**
148	SIVAPOOMATHI	30598	G2P1L1	21	h/o Macrosomia, PIH	45	145	21.4	108	101				
149	LAKSHMI	12347	G4P3LO	27	h/o Sudden IUD	58	149	26.1	152	123	93	170	122	102

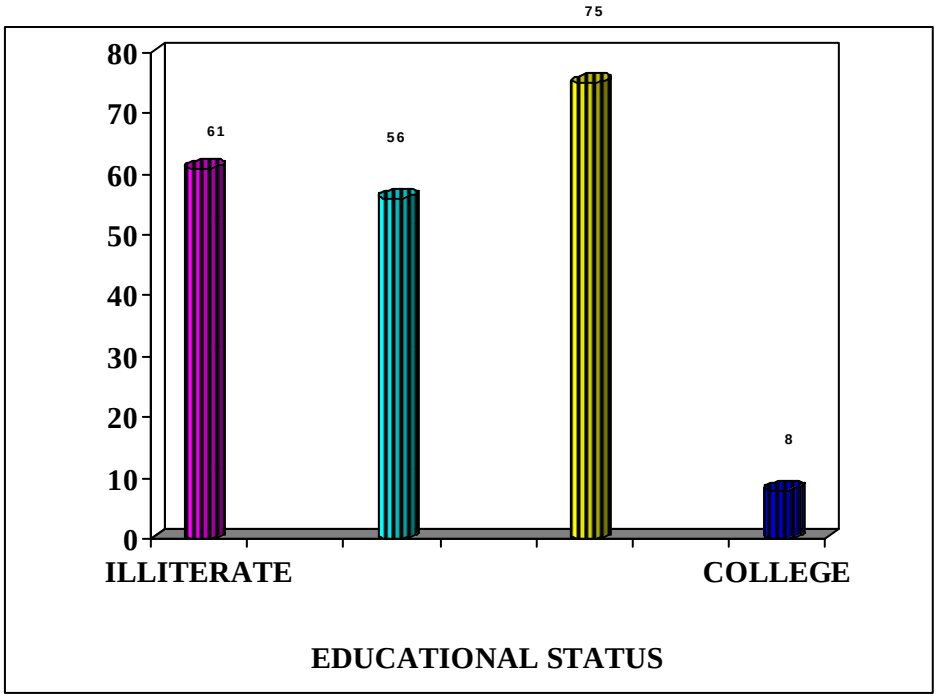
150	SELVI	31188	G2P1L1	24		45	142	22.3	87	82				
151	NAGALAKSHMI	93269	Primi	26	Hydramnios, Mother Diabetic	40	150	17.8	107	97				
152	JYOTHI	11700	Primi	23		56	156	23.0	112	104				
153	TAMILARASI	12514	G2P1L1	23		50	152	21.6	106	96				
154	VIJAYALAKSHMI	6211	G2P1L1	26	Heart Diseases Compl, h/o Outlet Forceps	63	150	28.0	109	101				
155	MAHALAKSHMI	10816	G2P1L1	22		53	150	23.6	102	92				
156	SATHYA	10841	G2P1LO	20	Hydramnios, Father Diabetic, h/o neonata death	53	143	25.9	94	96				
157	AYISHA	8290	G2P1L1	23		50	150	22.2	80	72				
158	ESWARI	7267	G4P2L1A1	25	h/o Abortion, Epilepsy Compl	48	148	21.9	115	105				
159	ISWARIYA	30198	Primi	21		55	153	23.5	118	102				
160	SOBHIA	30188	Primi	24	Anaemia	50	158	20.0	98	90				
161	CHELLAMAL	11729	G2P1L1	30		52	151	22.8	109	96				
162	JAYALAKSHMI	12526	G2A1	23	h/o Abortion	40	154	16.9	95	90				
163	VASUKI	12525	Primi	20	Glycosuria	40	162	15.2	136	122	80	120	76	70
164	MALIGA	12523	Primi	22		35	148	16.0	82	80				
165	GOMATHI	12525	Primi	26		45	155	18.7	71	82				
166	KOWSALYA	12527	Primi	19	PIH	45	151	19.7	108	101				
167	MANOHARI	5972	Primi	32		55	144	26.5		72				
168	BOWSIYA BEGAM	8738	G3P2L2	29		62	156	25.5	92	90				
169	THAVAMANI	12278	G2P1L1	24	Glycosuria	62	152	26.8	110	101				
170	SIVAGAMI	6536	G4P1L1A2	25	h/o Abortion	35	148	16.0	69	62				
171	BHAVANI	10860	G2A1	26	h/o Abortion	46	150	20.4	98	96				
172	BHUVANESWARI	12529	Primi	23	PIH	38	150	16.9	150	120	84	162	115	102
173	ANUSUYA	12530	G2A1	27	h/o Abortion, Hydramnios, Glycosuria	42	152	18.2	124	116				
174	KRISHNAVENI	12534	G2P1L1	29		55	151	24.1	116	112				
175	KALA	12538	G3P1L1A1	25	h/o Abortion	50	148	22.8	104	94				
176	MAYIL	12539	G2A1	20	h/o Abortion	55	152	23.8	102	92				
177	VIMALA	8553	Primi	19	PIH	40	148	18.3	99	94				
178	ESWARI	12532	Primi	19	Hyramnios, Congenital Anomaly	42	154	17.7	92	88				

179	USHA NANDHINI	11579	G2P1L1	24		73	158	29.2	68	74				
180	PANDEESWARI	12543	Primi	22		38	146	17.8	108	110				
181	RANI	12550	Primi	28	Mother Diabetic	44	151	19.3	102	98				
182	SELVI	12548	G2A1	20	h/o Abortion	48	160	18.8	88	86				
183	SUNDHARESWARI	12547	Primi	22		40	156	16.4	98	92				
184	SIVANDEESWARI	12546	Primi	27		46	151	20.2	112	101				
185	BANU	8745	Primi	23		35	147	16.2	101	86				
186	KALEESWARI	11778	G3P1L1A1	23	h/o Abortion, h/o Macrosomia	55	156	22.6	119	106				
187	SUMATHI	9274	G3P1L1A1	33	h/o Abortion	32	150	14.2	101	92				
188	KRISHNAVENI	12563	Primi	21		56	164	20.8	70	68				
189	SELVI	12634	G3P2L2	25		40	142	19.8	107	103				
190	PANCHAVARNAM	12635	G2P1LO	22	h/o neonatal death	35	150	15.6	113	103				
191	MEENA	12644	G2P1L1	23	h/o Macrosomia	42	142	20.8	109	102				
192	SUDHA	11665	Primi	23	PIH	42	152	18.2	120	110				
193	DURGADEVI	11427	Primi	20		42	147	19.4	97	88				
194	PANDISELVI	12129	G2P1L1	27		42	154	17.7	119	111				
195	INBADURGA	2469	G2P1L1	27	h/o Previous PIH	56	156	23.0	86	82				
196	USHA	9434	G2P1L1	27		40	143	19.6	113	103				
197	SASIKALA	31067	Primi	23		40	147	18.5	122	120				
198	OMSAKTHI	9992	Primi	20	Mother Diabetic	45	148	20.5	120	115				
199	VANITHA	4421	G3P1LOA1	24	Father Diabetic, h/o Abortion, h/o Outle Forceps	56	161	21.6	106	99				
200	RAJADEIVAKANI	12472	G2A1	26	PIH, Hyramnios, Congenital Anomaly, h/o Abortion	45	156	18.5	97	92				

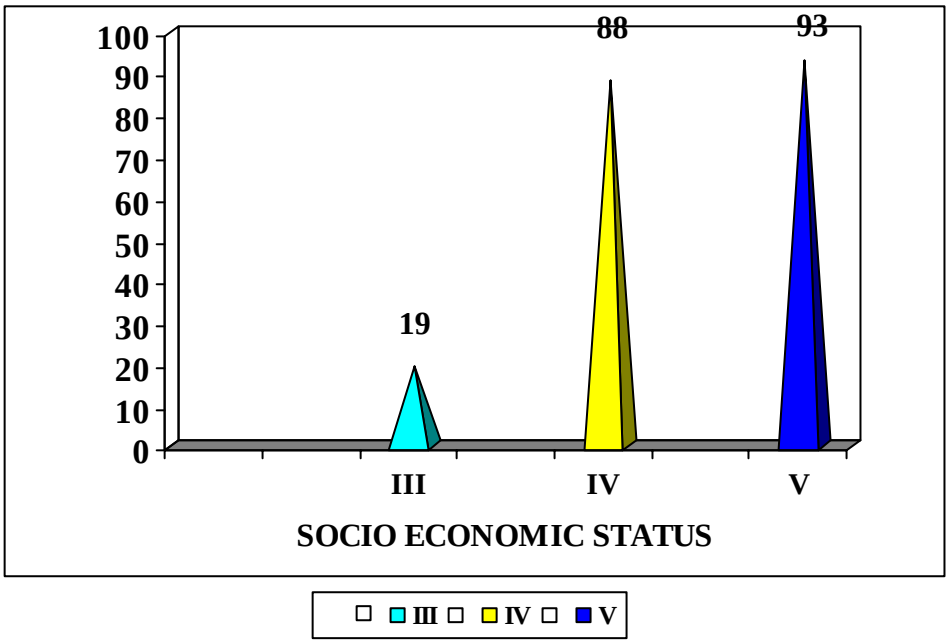
AGE DISTRIBUTION



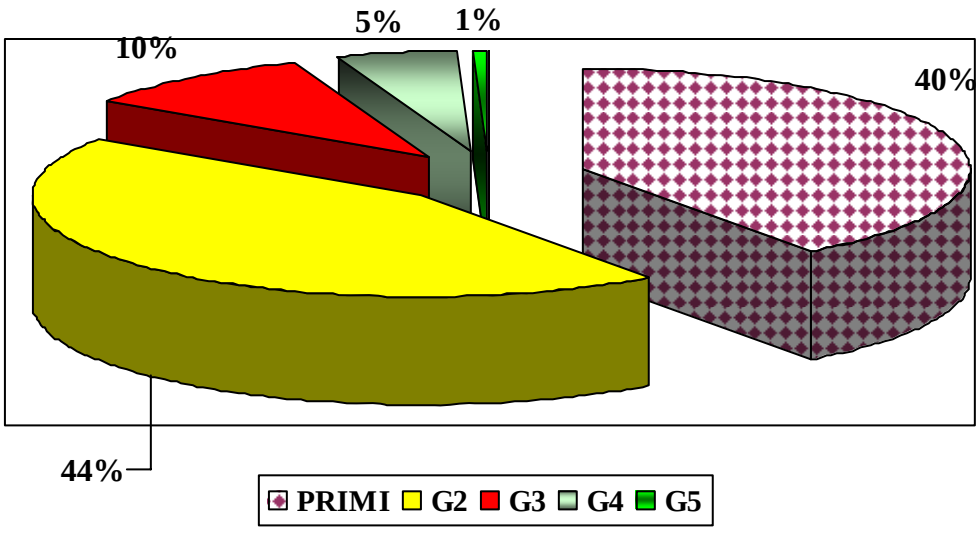
EDUCATIONAL STATUS



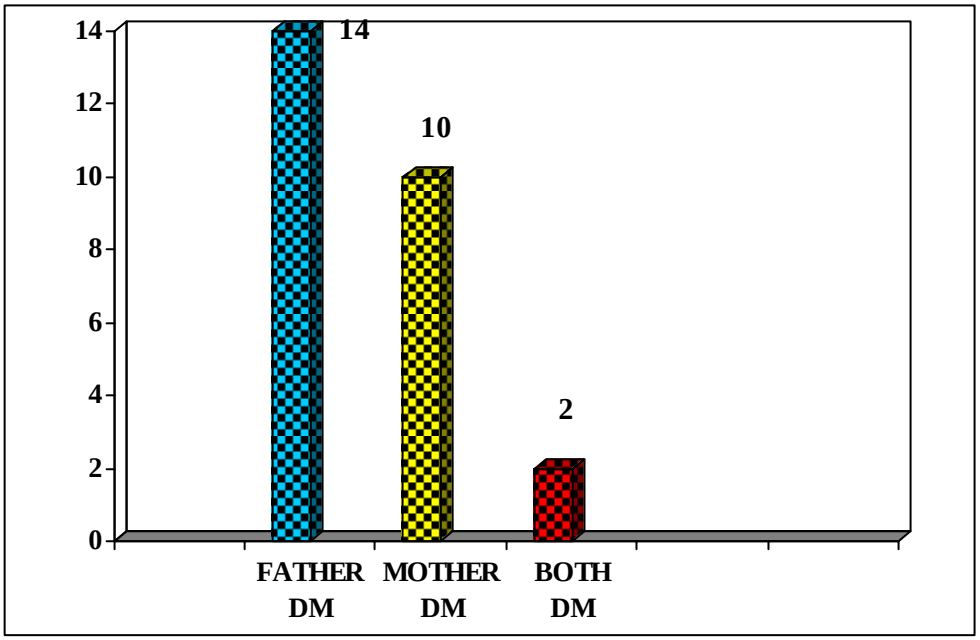
SOCIO ECONOMIC STATUS



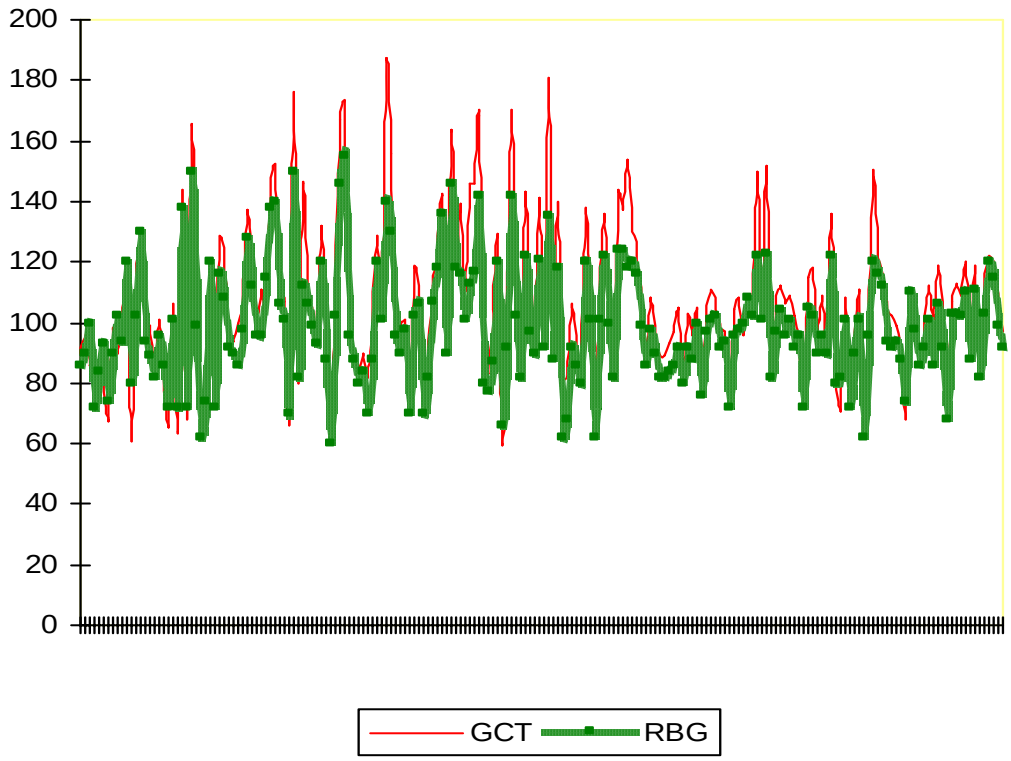
PARITY



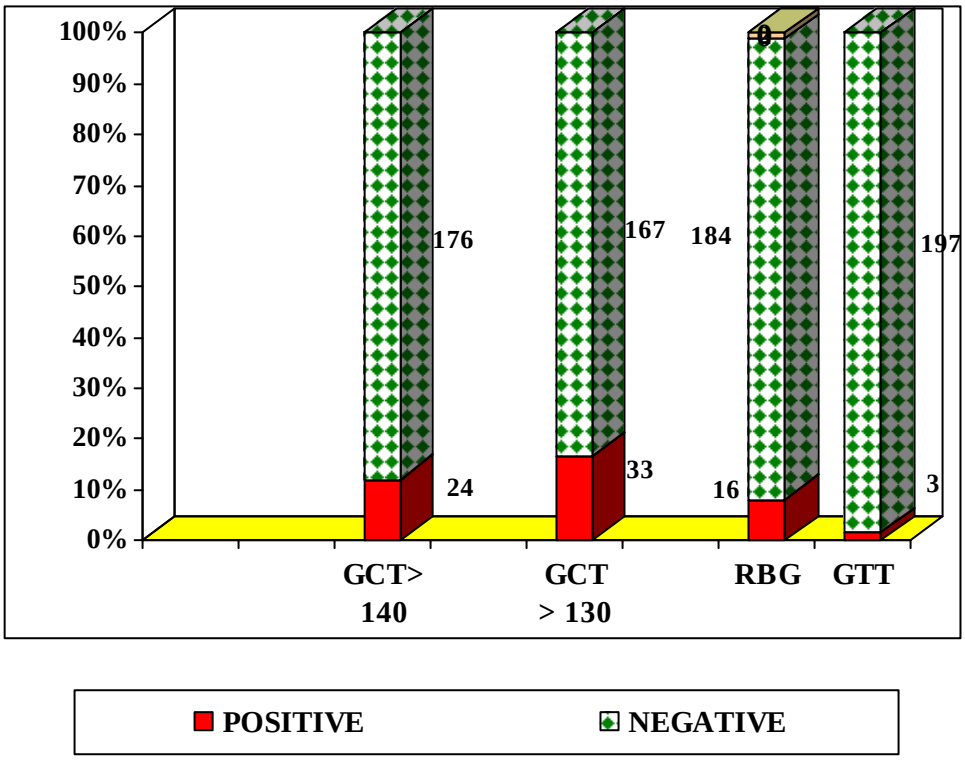
FAMILY HISTORY OF DM



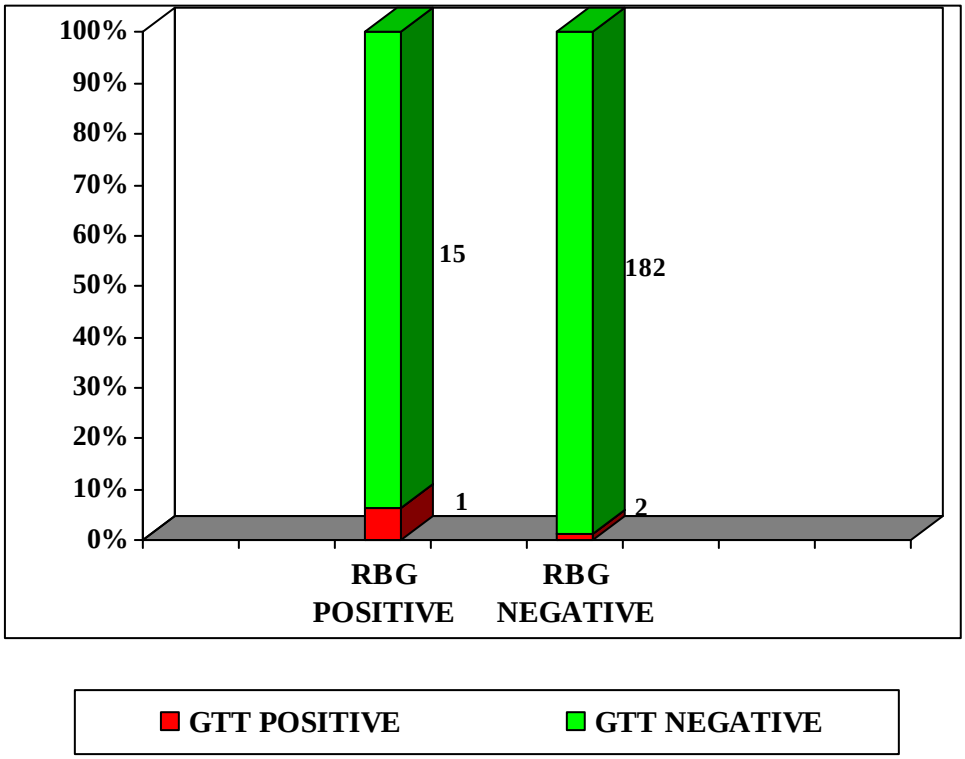
BLOOD GLUCOSE LEVELS
AS PER GCT & RBG



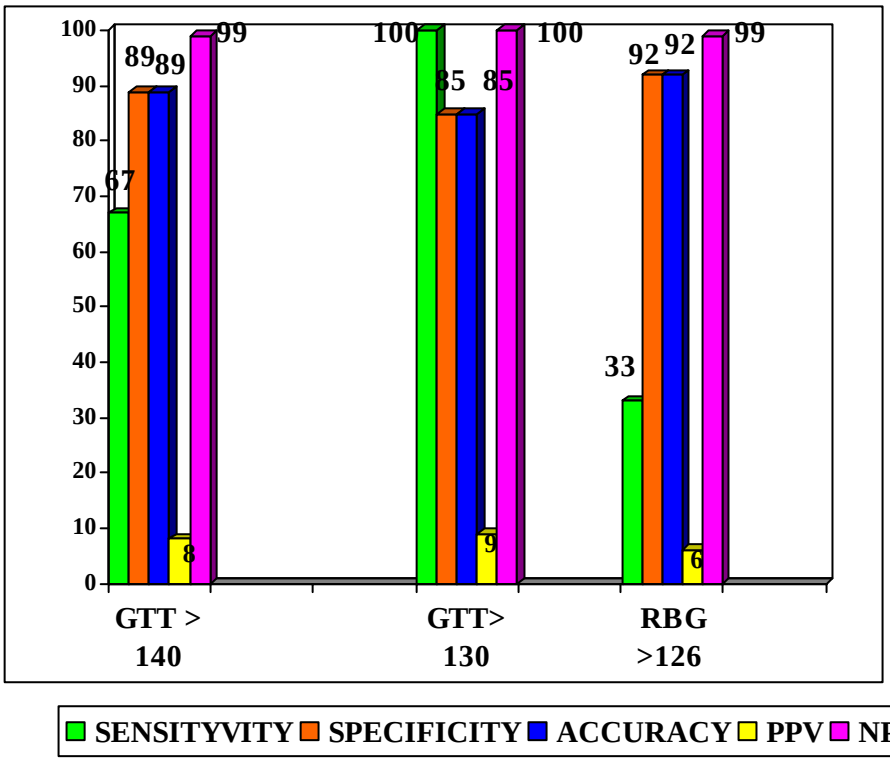
RESULTS OF VARIOUS TESTS



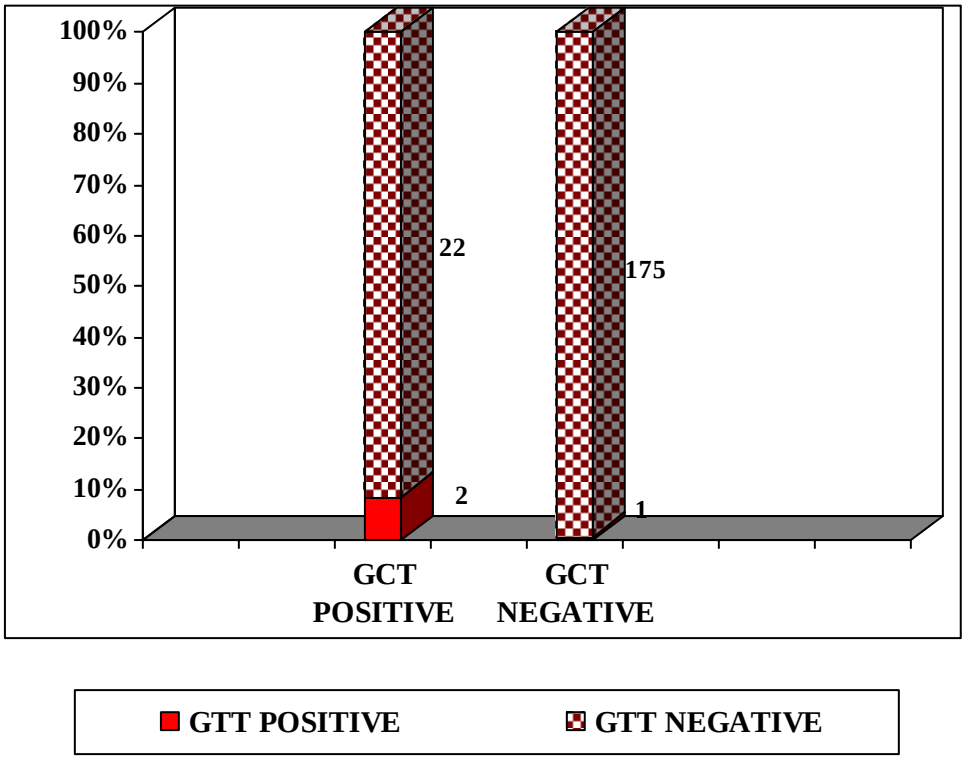
RANDOM BLOOD SUGAR > 126 AND GTT



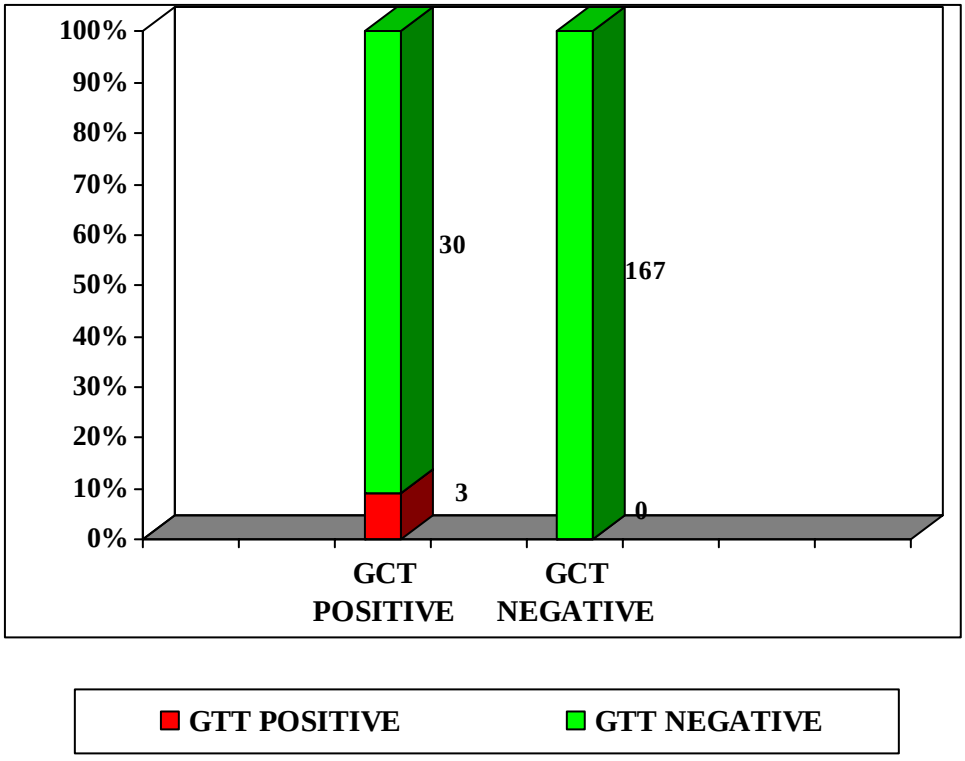
EFFICACY OF VARIOUS TESTS



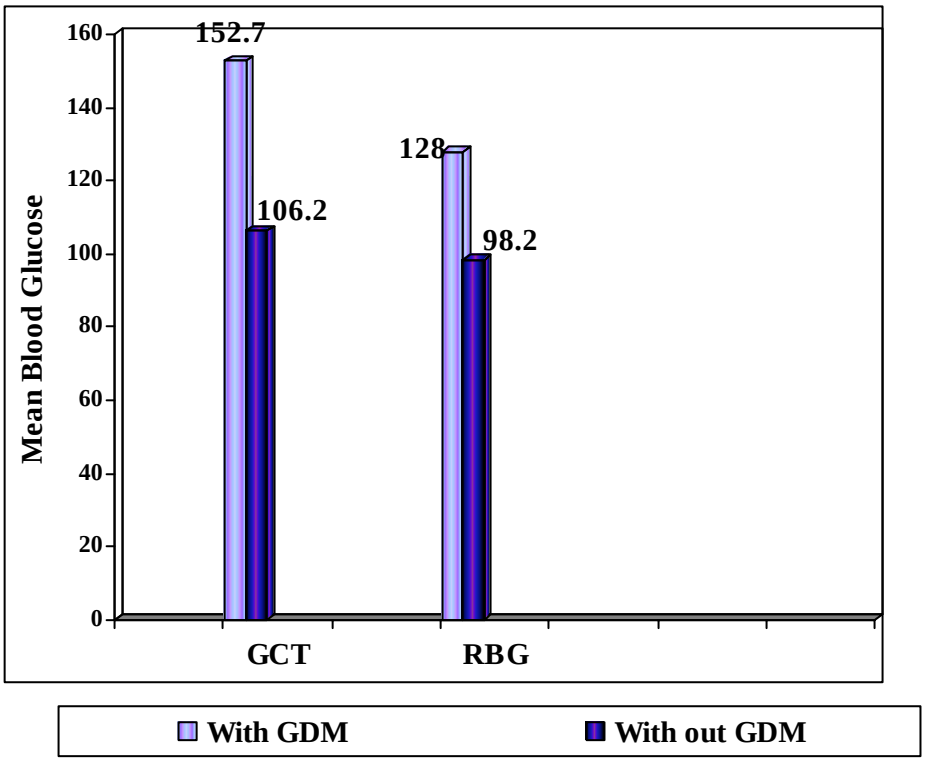
GCT > 140 AND GTT



GCT > 130 AND GTT



BLOOD GLUCOSE
AS PER GCT & RBG



GDM & AGE, WEIGHT & BMI